A TREATISE

ON

NERVOUS AND MENTAL DISEASES,

FOR STUDENTS AND PRACTITIONERS OF MEDICINE.

BY

LANDON CARTER GRAY, A.M., M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE NEW YORK POLYCLINIC; VISITING PHYSICIAN TO ST. MARY'S HOSPITAL; NEUROLOGIST TO THE HOSPITAL FOR RUPTURED AND CRIPPLED; EX-PRESIDENT OF THE AMERICAN NEUROLOGICAL ASSOCIATION; EX-PRESIDENT OF THE NEW YORK NEUROLOGICAL SOCIETY; EX-PRESIDENT OF THE NEUROLOGICAL SECTION OF THE NEW YORK ACADEMY OF MEDICINE; EX-PRESIDENT OF THE SOCIETY OF MEDICAL JURISPRUDENCE; HONORARY MEMBER OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA; FELLOW OF THE ACADEMY OF MEDICINE; MEMBER OF THE NEW YORK COUNTY MEDICAL SOCIETY; NON-RESIDENT MEMBER OF THE KINGS COUNTY MEDICAL SOCIETY, ETC.

SECOND EDITION, REVISED AND ENLARGED.

WITH 172 ILLUSTRATIONS AND 3 COLORED PLATES.

PHILADELPHIA:

LEA BROTHERS & CO.

1895.

PHILADELPHIA:
DORNAN, PRINTER.

PREFACE TO THE SECOND EDITION.

THE very kind reception accorded the first edition of this work has been a pleasant stimulus in my endeavor to make the present issue still more acceptable to students and practitioners. With this purpose in view, I have subjected the book to much alteration. Every portion has been carefully revised and five new chapters have been added, viz., those on Dementia, Dementia Paranoides, Confusional Insanity, Delirium, and Massage. The Anatomical Introduction has been entirely rewritten in response to many requests for a more detailed description of this subject, and also in view of the fact that our knowledge of cerebral and spinal architecture has been literally revolutionized in the last few years. In the chapter on Paralytic Dementia the pathological portion has been rewritten to conform with the newer views that are based partly upon the discoveries of Golgi and Cajal in cerebral histology, and partly upon our wider knowledge of textural stains. The chapter dealing with Palmus is almost new, and details my further experience with this disease.

Believing most thoroughly in medicine as a practical science, I have endeavored to mould this work so as to equip its readers with the knowledge necessary to the recognition and the most efficacious treatment of the diseases embraced in its title. The therapeutical sections were developed, with full detail, in the first edition, and they have been enlarged wherever advisable in the present issue. In every case I have recommended only those procedures which have been tested by experience. The word "treatment" has been construed in the broadest sense to include not only medicinal and non-medicinal agents, but also those hygienic and dietetic measures which are often the physician's best reliance.

I have deemed myself sufficiently familiar with the uses and abuses of Massage to devote a short chapter to the subject; but I have not treated of Hydrotherapy in its applications to nervous and mental disease because I am not yet convinced either that it is practicable outside of

a few large cities, or that it has more value than mere cleanliness. To obtain space for these additions and alterations without expanding the volume beyond the size best adapted to the student and physician, I have reluctantly omitted the bibliographical references, solacing myself with the thought that those possessing or having access to the first edition could readily obtain in the various annuals the literature of the two years which have intervened since its publication. The Glossary has been enlarged, in response to a general demand; and the Index has been made more complete. The series of illustrations has likewise undergone thorough revision. A comparison of the new edition with the first issue will show that many of the excellent engravings in the original have been completely re-executed in order to secure greater mechanical perfection in detail. All the cuts have been scrutinized, many have been redrawn, others altered, and I have added considerably to their number. The three colored plates which have been introduced in this edition will elucidate important facts in neural anatomy, physiology, and localization with a clearness unattainable with black alone.

L. C. G.

6 East Forty-ninth Street, New York,
September, 1895.

PREFACE TO THE FIRST EDITION.

This book is an endeavor to put a working knowledge of nervous and mental diseases into the hands of students and practitioners. A mere compilation, even of an encyclopædia, would have been comparatively an easy task, calling only for a judicious aggregation of material. In contradistinction, the purpose and limits of a treatise require the selection and clinical verification of facts, and to this most important element of my work the leisure of seven years has been devoted, only one of which, however, has been required for the actual composition. A teacher must know what to omit, as the force and permanence of his teachings will depend upon his strict avoidance of non-essentials. Superfluous knowledge, like surplus food, may clog the activity of the mind, and in no department of medical science is the temptation to expansion and speculation more enticing than in neurology. Keenly appreciating the patient toil of the scientists to whom we owe most of our knowledge, I have held the task of the physician in yet higher esteem, as the latter must make utilitarian application of all the facts collected by the former. There can be no loftier aim in medicine than the relief of human suffering, and to this end I have endeavored to glean from the many departments of medical science what they could contribute. Whether Americans are more prone to nervous disease than other races, or whether it is that our democratic principles set a higher value upon the individual, his comfort and health, than is customary elsewhere, it is yet certain that Europeans, in investigating disease, regard the patient simply as its vehicle, whilst Americans go one step further, and deem the cure all-essential.

Moulded by this conviction, this volume is addressed especially to the general practitioner, present or future, and its theme is rigidly therapeutical. Medical Nihilism is an error of youth and a confession of impotence, for Nature rarely afflicts man beyond the hope of relief. The therapeutist has enormous resources. Not only does the physical world provide a great variety of medicinal substances, and transmute its forces to suit his will, but likewise the psychical world is bounteous in its aid. Far from spurning his inheritance, the ideal physician should gather his knowledge of disease and its antagonists from every quarter, and exert every faculty for his patient's welfare, which is his own true success.

Those who may use this work will find that it does not assume previous knowledge of its subjects on their part. The Anatomical Introduction makes plain all the facts of structure necessary to a comprehension of nervous disease. The Electrical Chapter is entirely practical, even in the details of selection and use of batteries. The space assigned to the several diseases is determined by the needs of the profession at

large, a consideration which has made the chapter on Neurasthenia the largest in the book. If apparently undue space is devoted to Syringomyelia, Acromegaly, and Myxœdema, this is owing to the general interest in these new types, coupled with the lack of information apt to be shown about them even by specialists. Mental diseases are considered from the standpoint of the general physician, since the outcome will often depend on his skill in early recognition and treatment. I have endeavored to indicate what diseases of the mind may be best treated at home, what may need both home and asylum treatment, and the period of committal, and what may require seclusion from the outset. The incontrovertible fact has been generally ignored that mental diseases, like hepatitis or nephritis, are affections of a viscus, and the result has been a specialty within a specialty, and the relative disregard of the usefulness of the general practitioner. A novel feature in a work of this kind is the inclusion of the medico-legal aspects of nervous and mental diseases. I have presented this subject from the standpoint not of the lawyer but of the physician; and furthermore, have endeavored to qualify the reader as a medical judge of both sides of the questions at issue, a faculty of much greater value than an ability limited to the mastery of only one side in the capacity of a medical advocate.

In carrying out the therapeutic aim of the book, each chapter has been made to contain its own section on treatment. Especial care has been taken to make the therapeutical suggestions sufficiently detailed and precise to cover the varying stages, symptoms, and complications of disease, as well as to follow the important indications afforded by differential diagnosis. In the chapters upon Mental Diseases the most approved treatment is stated, and likewise the results to be expected. Only that knowledge has been admitted to these pages which has stood the test of experience.

Students will find the terminology rendered easy of acquisition by the derivations and definitions given in the Glossary. Throughout she volume simple Anglo-Saxon terms and familiar synonyms have been preferred. The limits of the book have rendered necessary a certain degree of terseness, but those who are desirous of further investigation will find a full bibliography appended to each chapter. I have made liberal use of illustrations, deeming them essential for explanation and vividness. They are largely original, being mostly made from specimens and photographs.

Many willing hands of kind friends have aided me in the great, often harassing labor of this book. To Dr. R. S. Newton I am indebted for several sections and brain specimens, and a photograph of a case of acromegaly. Dr. W. B. Pritchard assisted in the preparation of a tabulated statement of clinic cases. Dr. R. C. F. Coombes kindly furnished me with photographic portraits of typical cases of insanity.

6 East Forty-ninth Street, New York,
November, 1892.

CONTENTS.

PART I.

INTRODUCTORY.

CHAPTER I.

CHAPTER II.

CHAPTER III.

PART II.

NERVOUS DISEASES.

CHAPTER I.

CHAPTER II.

CHAPTER III.

CHAPTER IV.

CHAPTER V.

CHAPTER VI.

CHAPTER VII.

CHAPTER VIII.

CHAPTER IX.

CHAPTER X.

CHAPTER XI.

CHAPTER XII.

CHAPTER XIII.

CHAPTER XIV.

CHAPTER XV.

CHAPTER XVI.

CHAPTER XVII.

CHAPTER XVIII.

CHAPTER XIX.

CHAPTER XX.

PART I.

INTRODUCTORY.

CHAPTER I.

ANATOMICAL INTRODUCTION.

It is not my intention to write a text-book upon the anatomy of the nervous system. My purpose is only to make clear such facts as may be necessary to an intelligent comprehension of the clinical phenomena of nervous and mental disease; and in carrying out this purpose I shall not dwell upon abstruse matters that the future may show to be of great value, but shall rather confine my attention to the living issues of the day.

The nerve-cell is the essential element in nervous disease. In it go on all the peculiar molecular processes which endow the nervous system with individuality. It is the very inner citadel of nervous life. To it all the other textures are subsidiary. It is nourished by the arterioles and lymphatics, drained by the venules, upheld by the connective tissue. These cells of the nervous tissue have been very imperfectly known until the last few years, when the wondrous stain first suggested by Golgi made it possible for us to see into this hitherto hidden realm as no histologist had ever seen before. It was in 1886 that the Italian author described a method of coloring nerve-cells and their processes with the bichromate of silver, by placing them in a nitrate of silver solution after they had been hardened in bichromate of potash. In this way the cells and their processes are outlined as distinctly as in an etching. To obviate the difficulty of following these processes through the huge sections that were made necessary by the size of the human brain, the genius of Cajal hit upon the device of employing the brains of such small animals as the frog, the mouse, the rat, and the guinea-pig; and the results thus obtained, although met at first with the usual contradiction that seems to be the fate of all original observations, were soon confirmed at every hand. The result has been that the last few years have brought to our knowledge more exactness in regard to the histology of the nervous system than all the preceding countless centuries of time. Dictum after dictum has been ruthlessly brushed aside, purblind theory after purblind theory has shrunk to mere nothingness in the blaze of the light of truth that has so suddenly

come upon us; and, indeed, it is almost with a blush of shame that a neurologist, reading the latest work of Cajal, sits at the feet of this one man, who, with his followers, has penetrated one of the mysteries of the ages. It had long been known, from the time of Deiters and Wagner, that each nerve-cell had two sets of processes—namely, one known as the *axis-cylinder process*, supposed to be straight and unbranching, and the others known as the *protoplasmic processes*, which break up into numerous branches soon after leaving the body of the cell. The axis-cylinder process becomes continuous with the axis-cylinder of a nerve. The nature of the protoplasmic processes was unknown, some investigators maintaining that they served to connect the cells with one another, whilst others claimed that they split into very fine nerve-fibres, these ultimately serving the same purpose as the axis-cylinder processes. In this condition our knowledge remained stationary for several decades, until Golgi demonstrated that the axis-cylinder process was not straight and unbranching, but that it really gave off a number of very fine *collaterals*, and that the protoplasmic expansions, instead of forming a fine network, terminated in free extremities, after having given off numerous branches in their course. He maintained that the axis-cylinders passed into a network formed by the intermeshing of their collaterals. Cajal, however, has shown that the axis-cylinder processes all terminate in a free end, making connection with the terminal structure by *contiguity*, and not, as has been hitherto supposed, by *continuity;* and that the function of the protoplasmic processes is, in all probability, to conduct the nervous current into the cell. The nervous current, therefore, flows out of the cell through the axis-cylinder processes, and into the cell through the protoplasmic ones, being *cellulifugal* in the former and *cellulipetal* in the latter. Waldeyer has suggested the word "neuron" for the nervous unit that is composed of the nerve-cell, the axis-cylinder and its collaterals, and the protoplasmic process and its collaterals; and it is probably the patness of this term that has caused it to be almost universally adopted. The nervous system is made up of such neurons. By means of them the cell, couched far back in the screened depths of the nervous organs, is brought into direct and material communication with such far-away tissues as the skin, the muscles, the eye, the ear, the tongue, and the other organs to which nerves go. If this distant tissue be intended for the reception of impressions—as is the skin, for example—the impression can travel inward to the cell along these protoplasmic processes. If the tissue be a motor one, as, for instance, the muscle, the cell can make it contract by means of the axis-cylinder processes and their collaterals. These processes are, as it were, prolongations of the nerve-cell into the different motor and sensory tissues of the body; and the motor and sensory tissues of the body are in their turn, so to speak, peripheral appendages of the nerve-cell. The cells are mainly contained in the gray matter, whilst the axis-cylinder processes or nerves run in bundles in what is known as the white matter. The physiological study of the nervous system consists, therefore, in ascertaining what the functions are of the different

masses of gray matter or cells, or, if this is not possible, of the nerves or white matter. In other words, the cells collected in the gray matter are the ones that endow the nerves with their different functions, and we may ascertain what these latter are either by means of experiment upon the gray matter or upon the nerves leading to and from the gray matter. The complexity of function of the nervous system is due to the great variety of these cells, and its complexity of structure depends upon the varying topography, according to the needs of the human economy, of the masses in which these cells and nerves lie. It must always be borne in mind, however, that a nerve must take its origin from a cell, and that this cell generally lies in a mass of gray matter. We can therefore understand why it is that the brain and spinal cord are made up of masses of gray matter, or cells, which are connected by means of the nerves, or white matter. The chief of these masses of gray matter are to be found in the cortex of the cerebrum and the cerebellum, their basal ganglia, in the horns of the spinal cord and in the nuclei of the cord, or upper enlarged end of the cord, which is known as the medulla oblongata or oblong spinal cord. We have painfully gained our knowledge of the structure and functions of the nervous system by numerous methods, as follows:

1. Stilling first suggested the method of *Continuous Sections*, as, for instance, of the spinal cord, so that the course of certain nerves and their relation to certain cells could be fairly traced.

2. The *Coloring* method, due to Gerlach, was based upon the law that certain chemical reagents and coloring matters would affect only certain tissues, such as the cells and nerve-fibres, whilst the other textures would be either slightly affected, or not at all.

3. The *Comparative Anatomy* method, used with so much success by Meynert and his scholars, owes its value to the fact that in many animals the development of peripheral organs is proportional to that of their central structures from which the nerves of the former have taken their origin. This procedure has also the incidental value of making plainer the structure of the cerebrum of higher mammals, by comparison with the less complicated brains of those lower in the series.

4. The *Embryological* or *Developmental* method has attained some brilliant results at the hands of Flechsig and his scholars. It derives its value from the fact that the nerve-fibres of different portions of the nervous system take on their covering of myelin at different periods of fœtal life, so that this medullary covering may extend through a whole system of fibres, marking them out clearly to the eye, whilst neighboring systems are as yet unmedullated.

5. Gudden employed the *Atrophic* method, which is really one of an arrest of development, proceeding upon the assumption that certain portions of the nervous system will remain in an incomplete, almost fœtal stage of development, when the central organs from which they receive their nerve-fibres are destroyed. By this method different portions or centres of the brain were artificially injured or destroyed in young animals, and then the degenerative lesions were

traced from the point of destruction, thus mapping out the tract of nerve-fibres which had begun there. It has been found, however, that this method is only of positive value, and not of negative—*i. e.*, we can only deduce the relationship between two parts of the nervous system when destruction of one leads to lesion of the other; but we cannot, on the other hand, deny such connection when destruction of the one does not affect the other.

6. Occasionally a *Congenital Arrest of Development, or Malformation*, has thrown great light upon the function of some part of the nervous system.

7. The *Pathological Anatomical* method, or that of secondary degeneration, first instituted by Türck, has been of very considerable value, and it rests upon the belief that the structural integrity of a nerve-fibre depends upon the structural integrity of the nerve-cell from which it takes its origin, so that lesion of the latter induces a degeneration of the former.

8. The *Physiological* method, or that of Vivisection, consists in irritation (as by electricity) of certain centres of the brain, cord, or cerebellum, or of destruction of them (as by section), with a corresponding excitation or paralysis of the nerve-fibres proceeding from the irritated or destroyed parts. This method has given us some of our most valuable knowledge of the nervous system.

9. The *Pathological Physiological* method is that whereby pathological lesions demonstrably destroy the function of certain parts of the nervous system, and enable us to draw conclusions as to the physiology of the parts that have been injured.

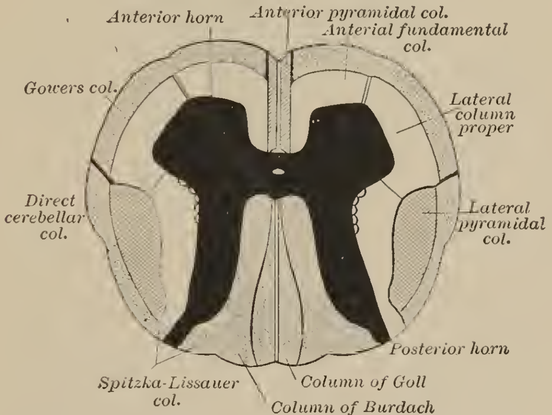
It is difficult to estimate which one of these methods has been of the most value. Suffice it to say, that from these various sources we have gained our knowledge of the anatomy of the nervous system, upon whose main features I now propose to dwell, so far as they are of clinical value.

We will begin with the spinal cord. In its study I shall not dwell upon the points of anatomy that are familiar to every medical student, such as the fact that the gray matter is disposed in the form of horns, those looking toward the belly, or *ventrad*, being the anterior horns or cornu, and those looking toward the vertebræ, or *dorsad*, being the sensory or posterior horns; whilst outside these horns or cornu of gray matter are grouped the nerve-fibres constituting the so-called white matter. As a matter of fact, therefore, the spinal cord consists in the main of a mass of nerve-fibres or white matter, enclosing the gray matter, or cornua. Each one of these nerves in the white matter is the conductor of some nerve impulse or function. To know what this function is has been the painful and eager study of neurologists, according to the various methods that have been just enumerated.

Fig. 1 is a diagram of the spinal cord, according to the knowledge that we possessed until a very recent period, and shows the motor columns outlined in white, the sensory being stippled, whilst the gray matter is seen in the centre colored in black. The motor columns were supposed to be the lateral and the anterior pyramidal

columns or pyramids, whilst the sensory tracts consisted of the columns of Goll, Burdach, Spitzka-Lissauer, direct cerebellar, and the Gowers. The fundamental column and the lateral column were very imperfectly understood at that period. This figure should be studied very carefully in order to obtain a clear comprehension of what is to follow; in other words, the names of the columns as they are found in this illustration are those which have been sanctioned by usage, and have so far maintained themselves in anatomical nomenclature, and, if they are thoroughly understood, it will be but child's play to memorize the modifications of this diagram that the latest investigations have made necessary. The student should therefore observe that the anterior pyramidal column is small, and is to be found on either side of the anterior median fissure; whilst the lateral pyramidal column is at the postero-lateral portion of the cord. These two columns, it may be said at this point, are the continuations of the motor fibres that have come down from the

Fig. 1.



Columns of the spinal cord, according to our late knowledge.

brain, the anterior pyramidal column containing those which have come down from the same side of the cerebrum, whilst the lateral pyramidal columns hold those hailing from the opposite hemisphere. The direct cerebellar column, it will be noted, is contained in the extreme postero-lateral periphery of the cord, external to the lateral pyramidal column; whilst the Gowers column is on the antero-lateral periphery of the cord, extending from the direct cerebellar column behind forward to the anterior pyramidal column. The column of Goll is a plume-shaped tract on each side of the posterior median septum. Outside of it, and between it and the posterior horn, is the column of Burdach. The Spitzka-Lissauer column is to be seen on each side of the posterior horn. This is generally known only as the Lissauer column, but as it was described simultaneously by Lissauer, of Berlin, and Spitzka, of New York, I have given it the compound name in this and my previous edition, as it

seems only just to do so. Fig. 2 will show the modifications of Fig. 1 that have been made by the so-called developmental studies of Flechsig—*i. e.*, by the assumption that a tract of fibres taking on its myelin at a different period from surrounding tracts is to be regarded as distinct anatomically and functionally. It will be observed that there are some important modifications in Fig. 2 over what has been represented in Fig. 1. For example, B is marked off from the anterior fundamental column, and is designated

Fig. 2.

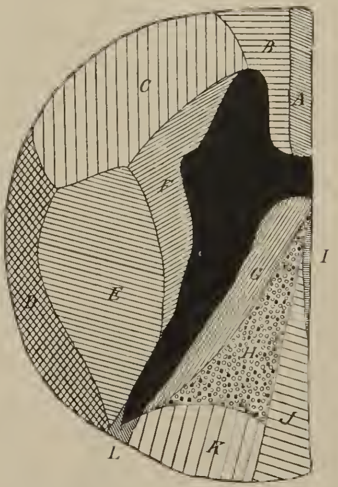


Diagram of spinal column, junction of dorsal with the cervical region, according to the Developmental Method of Flechsig.

A. Anterior pyramidal tract; *B.* Fundamental tract of the anterior column; *C.* Remaining lateral column; *D.* Direct cerebellar column; *E.* Lateral pyramidal column; *F.* Lateral limiting column; *G.* Anterior root-zone; *H.* Middle root-zone, composed of two systems of fibres, one being those of the first system, and the other being the second system; *I* Median zone of column of Goll; *J.* Column of Goll; *K.* Postero-internal root-zone; *L.* Postero-internal root-zone, or Lissauer's zone.

as the fundamental tract of the anterior column, whilst *C* represents the rest of the anterior fundamental and a part of the lateral column proper. Then, too, F is delimited from the lateral column proper, under the name of the lateral limiting column. It is, however, in the posterior columns between the diverging posterior horns that the most important modifications are to be found. For instance, the column of Burdach is split up into three different tracts, namely, G, the anterior root-zone; H, the median root-zone, the latter containing two systems of fibres; and K, the postero-internal root-zone. Nor is the column of Goll unchanged, as a portion of it (I) is delimited as its median zone.

Fig. 3 is a diagram of the spinal columns as they have been determined by secondary degenerations. At first sight this may appear very confusing, but it is exceedingly simple when it is

understood that all the fibres degenerating upward are sensory fibres, whilst all those degenerating downward are motor ones; in other words, all the fibres in which the degeneration is upward belong to a *neuron* whose cell is below the point of degeneration, whilst all the fibres that have degenerated downward are of a *neuron*

FIG 3

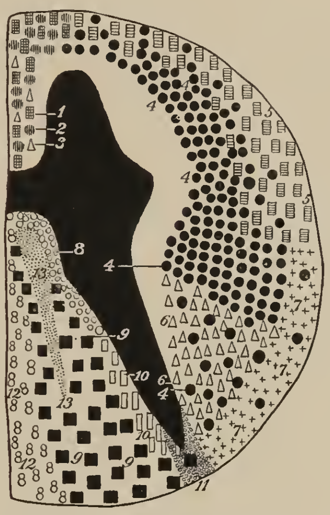


Diagram of columns of the cord, at junction of the dorsal with the cervical region, according to the Degeneration Method. (MARIE.)

A. Fibres Degenerating Upward. 1. Fibres of the sulco-marginal ascending tract; 5. Fibres of the Gowers tract; 7. Fibres of the direct cerebellar tract; 8. Fibres of the cornu-commissural tract; 9. Column of Burdach; 10. External root zone; 11. Zone of Lissauer; 12. Column of Goll.

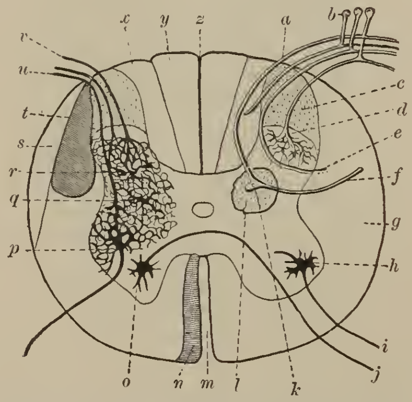
B. Fibres Degenerating Downward. 2. Sulco-marginal descending tract; 3. Direct pyramidal tract; 4. Intermediary tract of the antero-lateral column, some of these diverging and being found scattered in the Gowers column, the direct cerebellar column, and especially in the lateral pyramidal column; 6. Lateral pyramidal column; 13. Comma-shaped tract of Schultze.

whose cell is above this point. If this be clearly comprehended, the details of the figure become of exceeding interest, because they show that the columns of the cord marked out by the developmental method do not give an adequate idea of the way in which sensory and motor fibres of one column may intermingle, as, for example, in the intermediary tract of the antero-lateral column (4), some of which go into the Gowers column, others into the direct cerebellar column, and still others into the lateral pyramidal column; so that minute examination of the illustration will reveal the curious fact that the only pure motor columns are the anterior fundamental and the lateral, that the single absolutely sensory column is the Lissauer, and that the others are only mainly motor or sensory, and not exclusively so.

What a revolution has been recently undergone in our knowledge of the finer anatomy of the spinal cord can be ascertained by a care-

ful study of Figs. 4, 5, and 6. The first figure represents the views held until the researches of Cajal and Golgi were made, whilst the last three figures illustrate the discoveries of these authors and their confirmers. According to Fig. 4, the posterior root was composed of fibres coming from the columns of Clark and Burdach, the lateral column, and the posterior horn (*a*, *b*, *c*, *d*, *e*). The fibres of the column of Clark passed into the lateral column. The cells of the anterior cornua sent their motor fibres into the anterior root, which also received the nerve-fibres from the cornu of the opposite side (*i*, *j*). The protoplasmic processes of these same cells of the anterior cornua were supposed to form a network (*q*) which received the

FIG. 4.



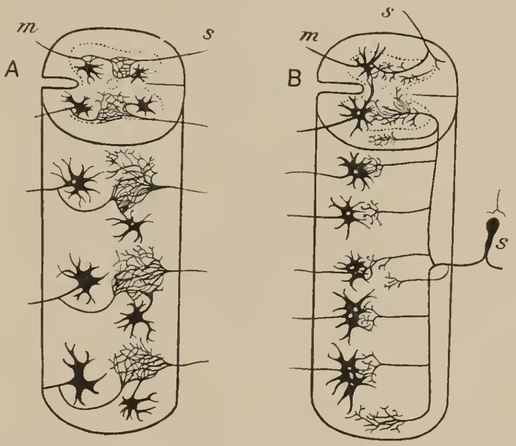
Connections of the anterior and posterior horns of the cord, according to the view formerly held. (Compare with Fig. 6.) (CAJAL.)

a. Posterior root-fibre originating in the cells of the column of Clark; *b*. Unipolar cells of the root-ganglion; *d*. Termination of a root-fibre in the network of the posterior horn; *e*. Root-fibre passing longitudinally across the lateral column; *f*. Fibre from the column of Clark to the lateral column; *g*. Lateral column; *h*. Root-cell continued in the fibre (*i*); *j*. Anterior root-fibre coming from a cell (*o*) of the anterior horn of the opposite side; *k*. Column of Clark; *m*. Anterior fissure of the cord; *n*. Column of Türck; *p*. Anterior root-cell whose protoplasmic branches form a network (*q*) where the posterior root-fibres terminate; *r*. Cells of the posterior horn whose protoplasmic processes form the network (*q*); *s*. Cerebellar column; *t*. Lateral pyramidal column; *u*, *v*. Posterior root-fibres terminating in a network; *x*. Column of Burdach; *y*. Column of Goll; *z*. Posterior fissure of the cord.

fibres from the cells of the posterior cornua (*r*), these same cells sending off their nerve-fibres to the posterior roots (*u*, *v*). Golgi, with his wonderful stain, showed that these views were incorrect, demonstrating that each axis-cylinder process of a cell gave off collaterals. He maintained that these collaterals from the cells respectively of the anterior and posterior horns formed the network of communication (Fig. A, 5). Cajal, however, showed an entirely different set of connections, which have been universally confirmed, and his views are illustrated in B of Fig. 5. From S the unipolar cell of a ganglion of the posterior root sends off a cellulifugal fibre to the spinal cord, through the posterior root.

When this arrives in the cord, it bifurcates into an ascending and a descending branch, both of which give off, at right-angles, fine horizontal collaterals that proceed to the motor cells of the anterior horn, surrounding them with arborizations. Even the trunk of the posterior root gives off collaterals to the cells of the anterior horn. Cajal has furthermore demonstrated that the nerve-cells are independent units, that anastomose neither with their protoplasmic processes nor with their axis-cylinder ones; that each axis-cylinder terminates by varicose and flexible arborizations similar to those in the motor plate of muscles; and that, most startling of all, these arborizations are applied either to the body or to the pro-

FIG. 5.



A. Opinion of Golgi regarding the communication of the anterior and posterior roots. *m*. Anterior root-fibre; *s*. Posterior root-fibre, both united by networks formed in the gray substance by the union of the terminal ramifications of the rearmost collateral of the anterior root-fibre (*m*) with those of the posterior root-fibre; in these networks are the arborizations of the fine axis-cylinder from the sensitive cells of the posterior horn.

B. Cajal's view of the relations between the posterior and the anterior horns. *s*. Unipolar cell of a spinal ganglion; *m*. Anterior root-fibre. The cellulifugal fibre of the spinal ganglion-cells, passing to the posterior root of the cord, at the level of the posterior column, bifurcates into an ascending and a descending branch, giving off at a right-angle fine horizontal collaterals which go to the motor cell of the anterior horn, surrounding them with their terminal arborizations. The trunk of the posterior root-fibre itself gives off a few collaterals in the same direction.

toplasmic processes of cells, and establish connection by *contiguity*, and not, as has been tacitly held since the study of anatomy began, by *continuity*. These two great discoveries of the collaterals and the connection by contiguity made waste-paper of all previous diagrams of the cord, and brought the oldest anatomist to a par of knowledge with the student. The study of spinal anatomy now became a study, not of nerves, but of collaterals—*i. e.*, the branches of nerves. These discoveries led in their train to the further fact that the cells of the gray matter of the spinal cord did not alone consist of those which supplied fibres to the anterior and

posterior roots, but that there were others which had no such connection, furnishing instead fibres to form the commissures of the cord or its different columns. Of these we shall have more to say soon. Fig. 6 is a diagram from Cajal, which gives the results of his latest studies. It will repay us to study this briefly. It will be seen that the collaterals of the column of Goll (*a*, *b*) go to the posterior horn, the posterior commissure, and even as far forward as the anterior horn (*c*). The posterior root-trunk has also its collaterals, as has already been said. Certain collaterals from the anterior columns take part in

Fig. 6.

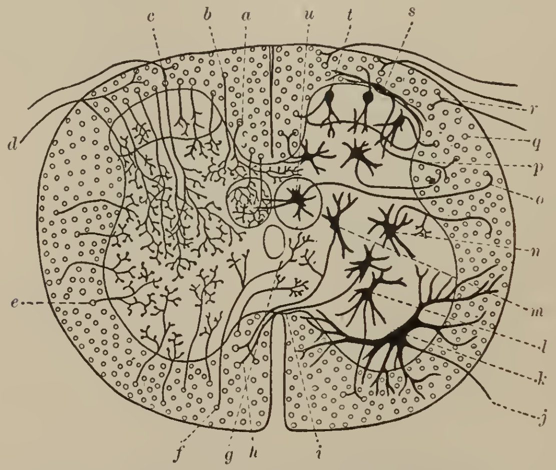


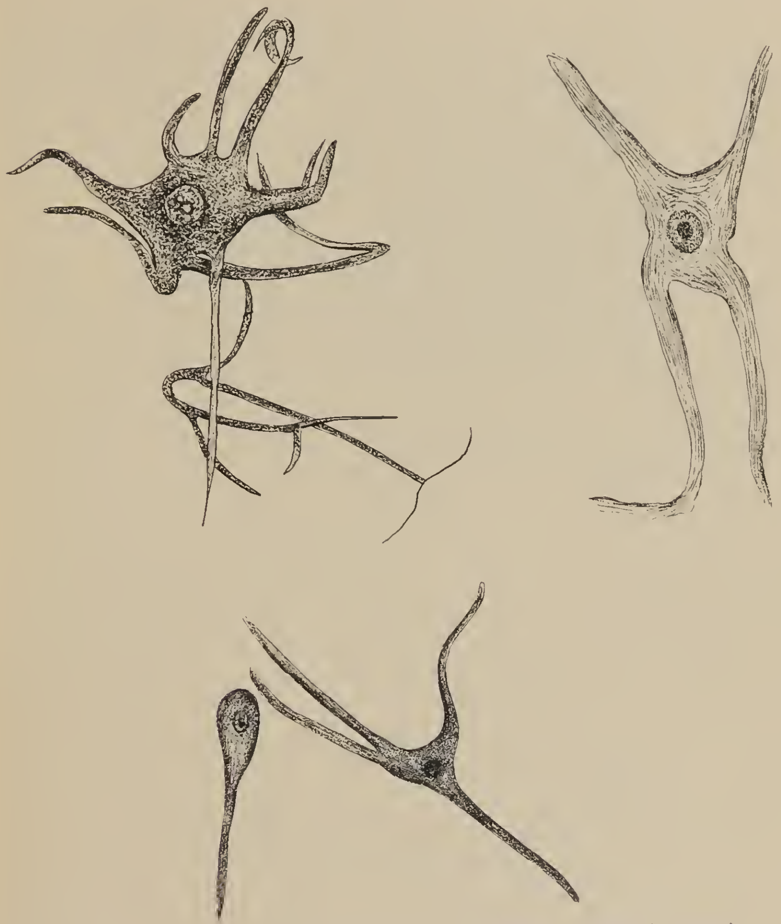
Diagram representing a section of the cord, showing the relation between its different elements according to the modern discoveries. (Cajal.)

a. Collaterals of the column of Goll forming a larger part of the posterior commissure; *b*. Collaterals of the same column going to the posterior horn; *c*. Collaterals of the posterior column reaching to the central gray substance and extending forward to the anterior horn; *d*. Posterior root-trunk and its collaterals; *e* Collaterals of the anterior column; *f*. Collaterals forming the anterior commissure; *g*. Their course in the commissure; *h*. Axis-cylinder coming from a commissural cell, and which, after having passed through the anterior commissure, goes to the anterior column; *i*. Course of this axis-cylinder in the commissure; *j*. Axis-cylinder going directly to the anterior horn, and coming from *k*, a large motor cell; *l*. Cell of the anterior column with a bifurcated axis-cylinder; *m*. Cell with a commissural axis-cylinder; *n*. Cell whose axis-cylinder gives off collaterals for connection; *o*. Axis-cylinder of a cell of the column of Clark; *p*. Axis-cylinder coming from *s*; *q*. Transverse section of an axis-cylinder; *r*. Bifurcation of the posterior root-fibres and ascending and descending branches; *s*. Marginal cell of the substance of Rolando; *t*. Small cell of the same substauce; *u*. Body of the cell of the column of Clark.

the formation of the anterior commissure (*f*, *g*). If the course of (*h*) is followed carefully, it will be observed that it terminates in the anterior column, after having crossed from the other side of the cord in the anterior commissure (*i*) and having had its origin in a so-called commissural cell. On the other hand, (*j*) shows that there are cells (*k*) giving off axis-cylinders directly to the anterior root. A column-cell is observed at (*l*), sending its bifurcated axis-cylinder to the anterior column. A commissural cell is at (*m*). At (*n*) is a cell whose axis-cylinder gives off collaterals for connection. The cells

of the column of Clark send their axis-cylinders to the lateral columns (*o*). The bifurcation of the posterior root-fibres into ascending and descending branches is seen at (*r*). At (*s*) is seen a marginal cell of the substance of Rolando, whose axis-cylinder is at (*p*). Another small cell of the Rolandic substance is at (*t*).

FIG. 7.



Drawing of cells from the anterior horns of the spinal cord. Obtained by tapping the fresh cord of an ox.

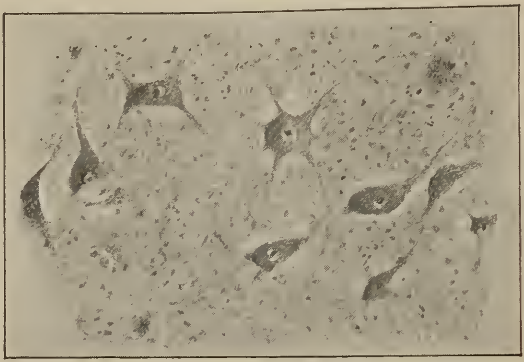
As has already been said, it used to be thought that the cells of the spinal cord were only of the one kind that gave origin to root-fibres, either posterior or anterior. The newer methods of staining, however, have shown that there are five varieties, viz.:

1. Root-cells;
2. Commissural cells;
3. Column-cells;

4. Many-column or pluricordonal cells;
5. Cells with a short axis-cylinder.

The first four varieties all lose their axis-cylinders in the white substance, so that they may be called cells with long axis-cylinders, whilst the fifth kind parts with its axis-cylinder within the gray matter.

FIG. 8.



Drawing from a photo-micrograph, made by McDONALD, showing the cells of the anterior cornua as they lie imbedded in a specimen hardened in bichromate by the alum Weigert method, and afterward magnified 360 diameters.

FIG. 9.

Motor cell of the anterior horn, from the spinal cord of a newborn dog. (CAJAL.)

We have first to obtain an adequate idea of the form and shape of these cells from the bichromate of silver staining. Our knowledge of them in olden times was obtained from tapping the

fresh spinal cord of an ox, when we obtained the shapes which are seen in Fig. 7. Then other stains employed with hardening methods enabled us to see these structures as they lay imbedded in the spinal cord, and we had such a view as is represented in Fig. 8, which shows a photo-micrographic drawing of a section magnified 360 times. How little idea we had of the vast complexity of these minute cellular bodies can be gathered from a comparison of these two figures with Fig. 9, which represents the motor cell of the anterior horn from the spinal cord of a newborn dog. An easy distinction can be made between the protoplasmic and the axis-cylinder processes. The former, as will be seen in Fig. 9, have many branches, which can be divided into antero-external, posterior, and internal divisions, each branching dichotomously; and the latter are colored in red.

FIG. 10.

Column-cells of the human spinal cord. (LENHOSSEK.)

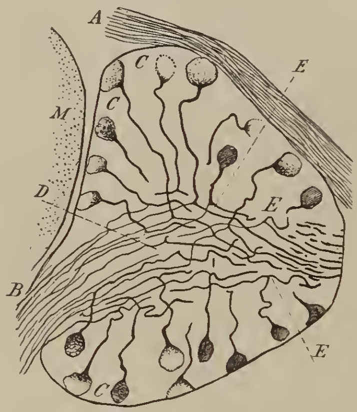
The column-cells are so called because from them arise the axis-cylinders which form the different columns of the cord. Most of them are contained in the anterior horn, and send their axis-cylinders into the antero-lateral column (Fig. 6, *n*, and Fig. 10). Those of the posterior horn have their axis-cylinder processes directed to the most posterior region of the lateral column. Others in the substance of Rolando and the internal portion of the posterior horn send fibres to the posterior column. In the column of Clark there are two varieties of cells—the commissural cells sending fibres to the anterior commissure, and column-cells whose fibres go to the lateral column,

probably to the direct cerebellar column. The latter sometimes pass off without division, and sometimes by what is known as the T-shaped division, splitting the original fibre into an ascending and descending vertical branch. The multiplicity of these column-cells is beautifully figured in 10.

The commissural cells (Fig. 6, *m*) are smaller, and have fewer processes than the root-cells. They are found in the thickness of the gray substance, and usually send their axis-cylinders through the white commissure to the antero-lateral column of the opposite side, sustaining at this latter point, however, the T-shaped division into an ascending and descending fibre.

The many-column cells are those whose axis-cylinder splits up in the gray matter into two or three fibres, which each go to a different column, so that one such division may pass into the anterior column of the same side, and the other division into the same column of the opposite side; or, on the other hand, one of these branches may enter the posterior column, and the other the lateral or anterior column.

FIG. 11.



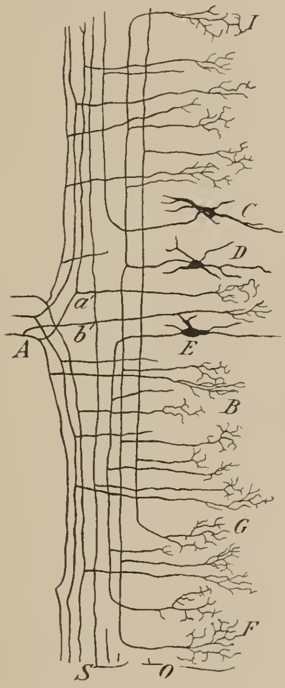
Section of posterior root-ganglion of a rat, eight days old. (CAJAL.)

A. Anterior root. *B.* Posterior root. *C.* Body of the ganglionic cell. *D.* Internal or central branches of the dichotomous division, *E*, of the nervous fibre of the ganglionic cells. *M.* Spinal cord.

Each posterior root not only contains sensory fibres, or centripetal, but also centrifugal or motor ones. The sensory fibres come from the cells of the ganglion upon the posterior root, as will be seen by studying Figs. 11 and 12. Each one of these ganglionic cells is enveloped in an endothelial capsule. Outside of this capsule—*i. e.*, between the capsule and the cellular protoplasm—is a very fine pericellular arborization continuous with a nervous fibre whose origin is yet unknown, so that this ganglionic cell receives, it would appear, not only a current through the fibre coming from the periphery and transmitted to the spinal cord, but also a nervous impulse coming from some unknown quarter, possibly from a cell of the sympathetic

system, as some of Cajal's researches would seem to show. It was at first supposed that each posterior root entering the posterior column passed into two divisions, one of which became vertical, passing up to the medulla oblongata and the brain in the white substance, whilst the other segment was lost in a nervous network formed by the anastomoses of the protoplasmic expansions of the cells, or those of the collaterals and the terminal branches of the axis-cylinders. (See Figs. 4 and 5.) In 1887, however, Nansen made the surprising discovery that in the *myxina glutinosa*, one of the lowest of the fishes, of the hag or sucker order, the posterior root-fibres bifurcated in the

FIG. 12.



Half diagrammatic longitudinal section of the posterior column parallel to the entrance of the posterior roots. (CAJAL.)

A. Posterior root; *S*. White substance; *O*. Gray substance; *C*. Cell of the posterior column with a curved axis-cylinder; *D*. Another cell of the posterior column with an axis-cylinder bifurcated into ascending and descending branches; *E*. Another cell with a curved descending axis-cylinder; *F*, *G*. Terminal arborizations of the axis-cylinders; *B*. Terminal arborizations of the collaterals of the white substance; *a'*. Collateral of the branches of a posterior root-fibre; *b'*. Collateral of the trunk of another posterior root-fibre.

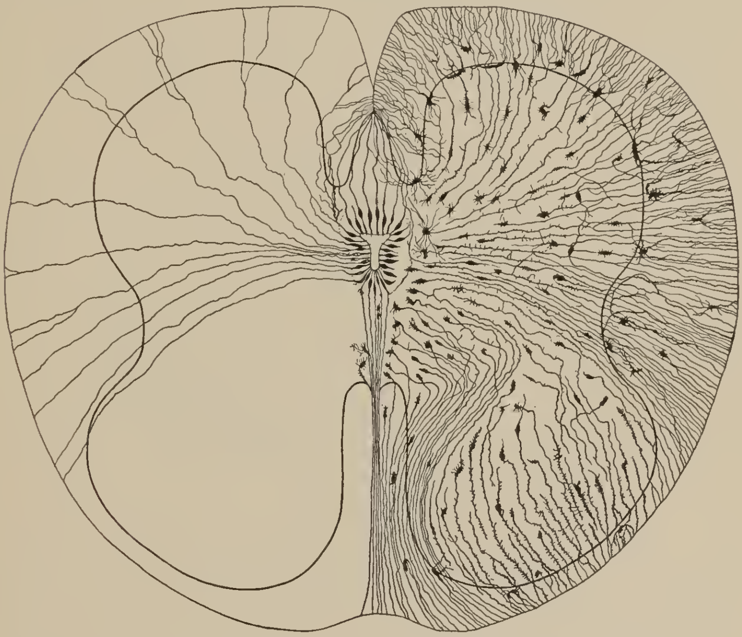
white substance, and that the branches of these bifurcations assumed a longitudinal direction and gave off collaterals for the gray substance. This arrangement was soon demonstrated by Cajal in birds and vertebrata. The centripetal fibres composing the bulk of the posterior root are of two varieties, one external or fine, the other internal or thick. These enter the posterior column obliquely, then bifurcate into an ascending and a descending branch, both of which are longitudinal, and both probably enter the gray substance after a course of several centimetres in the white substance, and terminate by free arborizations lying between the elements of the posterior horn and the substance of Rolando. (Fig. 12.) The external or fine tract of the posterior

root has its terminal bifurcations in the Spitzka-Lissauer column and the neighboring portion of the lateral column, and its collaterals are sparse, delicate, and appear to terminate alone in the posterior horn. The internal tract, or that of the thick fibres, passes to the column of Goll and Burdach, and it furnishes the greatest number of collaterals of the sensory-motor tract going forward to the cells of the anterior horn. From the trunk of the posterior root, as well as from its ascending and descending fibres, a number of fine collaterals pass off at a right angle, penetrating the gray substance, and there terminating by beautiful free arborizations in contiguity with the bodies of the cells in the posterior and anterior horn. The plan of entrance of these posterior root-fibres is shown in Fig. 12, *A* being the posterior root-fibre, *a′* a collateral which passes off from it, *b′* another emanating directly from the posterior root-trunk. *S* is the white substance. In it is seen a curved ascending axis-cylinder coming from a cell (*C*) in the posterior column, whilst from *D*, another cell of the posterior column, passes off an axis-cylinder which bifurcates into an ascending and a descending branch. *E* is still another cell of the posterior column, with a curved descending axis-cylinder. *F* and *G* are the terminal arborizations of axis-cylinders, whilst *B* represents the terminal arborizations of the collaterals of the white substance. A large number of the collaterals of the posterior roots reunite into an antero-posterior tract which crosses the posterior horn and expands, fan-like, around the motor cells of the anterior horn. This is the sensory-motor tract of Cajal, or the reflex-motor tract of Köllicker, and, as the name indicates, it is the material highway for such reflexes as those of the tendons.

The nervous elements are sustained by two kinds of corpuscles—namely, the epithelial cells, and the neurogliar, or spider, or Deiters' cells. The epithelial cells cover the cavities of the nervous centres. They are elongated bodies, with a nucleus, and with one or more ramifying and divergent processes which are lost in the neighboring white or gray substance. In the embryo these epithelial cells are very long, and are prolonged from the central cavity to the exterior surface beneath the pia mater, where they form cones, and thus constitute a sort of limiting membrane. (See Fig. 13.) In the brain of certain vertebrata, as in fishes, reptiles, and batrachians, this disposition is maintained throughout life, and in these organisms there is no other connective tissue. But in the spinal cord and the cerebrum of birds and mammifera this tissue atrophies, for the expanding processes do not reach to the surface of the nervous organ, although in the olfactory mucous membrane and the retina the embryonic conditions are maintained. The neurogliar cells—sometimes called the spider or Deiters' cells—are to be found in great abundance in the white substance of the nervous centres, in the framework of the nerves, in the ganglia of the sympathetic, and, in less quantity, in the gray cerebro-spinal masses. They are small, with very fine flexible or slightly ramified processes which terminate free, often by fixing themselves upon the surface of the capillaries. The connective tissue of the nerve-centres is the result of a plexiform inter-

crossing of the expansions of these neurogliar cells, but not of any anastomosing, as was formerly supposed, and the function of these expansions appears to be to sustain and isolate the medullated fibres. It is a matter of very great doubt, however, whether they also sustain and isolate the nerve-cells. The origin of these neurogliar cells has been the cause of a great variety of opinions. At the present day it is believed by most histologists that they are transformed epithelial corpuscles which have emigrated from their usual site upon the interior surface of the nerve-centres, and that this transmutation has been effected by atrophy of their central and peripheral processes and by the production of secondary ones. During this process of transformation the epithelial cells would seem to be capable of multiplying, according to Lenhossek. Fig. 13 shows upon the

Fig. 13.



Spinal cord of a human embryo prepared by Golgi's method. On the left side is the epithelial or ependymal structure; on the right, the neurogliar cells. (Lenhossek.)

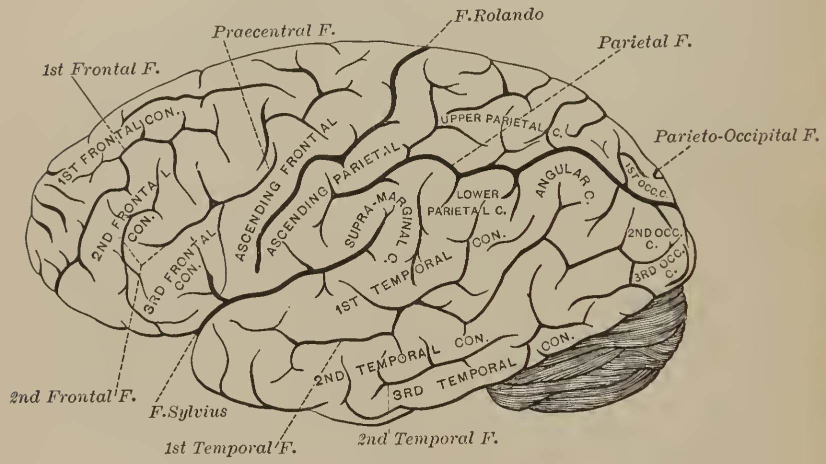
left side the epithelial structure, and upon the right the neurogliar cells. For a certain time the epithelial corpuscles preserve their peripheral process which passes to the pia mater; but gradually this atrophies, as well as the central process, and thus is effected a transformation of the epithelial cell into a neurogliar one. The epithelial cells, however, of the anterior and posterior fissures preserve their form and their primitive length.

So much for the spinal cord proper. Before entering into con-

sideration of the structure of its upper enlarged end, known as the medulla oblongata, it will be best to study first the brain and cerebellum, and then show the connection between these three structures.

I desire to say at the outset of this portion of my subject that all my drawings, unless otherwise indicated, were made from actual fresh brains, or relatively fresh ones that had been preserved for a day or two in a salty solution, or else from those hardened in alcohol, and in some cases photographs or silver plates have been made of the brain, and the drawings from these. In every instance, therefore, the convolutions are exactly as they are found in the brain. I am particular in calling attention to this point, because in scarcely one of the text-books is use made of such fresh or hardened brains, the representations being generally by means of diagrams. The result has been that most of the readers of books have become conversant with the convolutions only through such

FIG. 14.



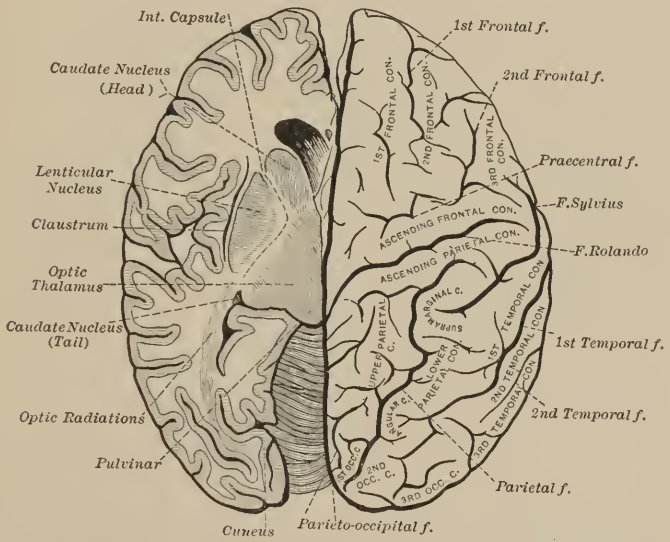
Convolutions of the left hemisphere.

diagrammatic representations, and I anticipate, therefore, that critics who are not familiar with actual brains will be surprised at the course of some of the convolutions. Even Meynert, Ecker, Edinger, and Schwalbe, who are certainly indubitable authorities upon cerebral anatomy, make use almost entirely of diagrams. The truth of the matter is that the convolutions vary so much in different brains that slight deviations from the usual course are of no importance, and it is very doubtful whether we know at the present time what really constitutes a pathological variation in the direction of a fissure.

The fissures of the brain are in the main very simple. In Fig. 14, which is the lateral surface of one hemisphere, it will be seen that there is a large fissure going upward and backward from the base of the brain. This is the fissure of Sylvius. Coming down from above, from the great longitudinal fissure, is another large fissure, known as the fissure of Rolando, almost running into the fissure of Sylvius.

These two fissures can easily be found in a brain by the facts that have already been stated, namely, that one runs up from the base, that the other runs down from the great longitudinal fissure, and that both are large. The fissure of Rolando is the boundary line between the frontal lobe in front and the parietal lobe behind; whilst the fissure of Sylvius is the boundary line between the frontal and parietal lobes above and the temporal lobe below. It will be noticed that the convolutions behind and in front of the fissure of Rolando are ascending, whilst the other convolutions of the frontal and parietal lobe pursue a more or less horizontal course. These two convolutions, therefore, one in front of the fissure of Rolando and the other behind it, are known as *the* ascending convolutions; and as the one in front belongs to the frontal lobe, it is known as the ascending frontal convolution, whilst the one behind, belonging to the parietal lobe, is called the ascending parietal convolution. In front of the ascending frontal convolution in Fig. 14 is seen a fissure which is more or less continuous from above downward, and which, like the fissure of Rolando, has an ascending direction.

Fig. 15.

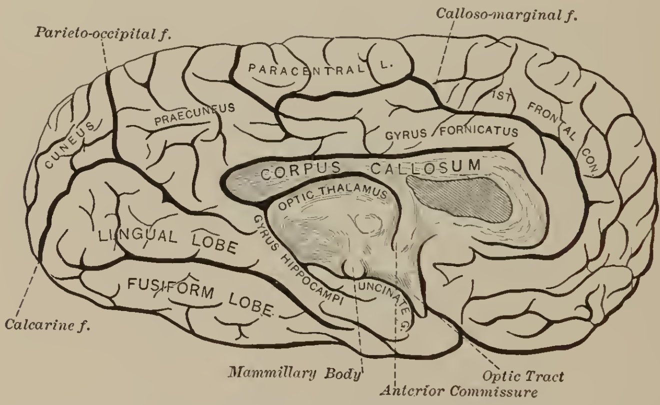


Convolutions of the vertex, on the right; on the left, the basal ganglia, internal capsule, centrum ovale, and cuneus.

This is an accessory fissure, the præcentral, and it is of no special importance except that its position must be borne in mind, so that it will not be confounded with the fissure of Rolando. It will be seen that in front of the ascending frontal convolution there are two main fissures curving forward. The upper one is known as the first frontal fissure and the lower as the second frontal fissure; and they serve to mark off this portion of the frontal lobe into the first frontal convolution, which lies above the first frontal fissure;

the second frontal convolution, between the first and second frontal fissures; and the third frontal convolution, below the second frontal fissure. The third frontal convolution, it will be observed, has a fork or anterior branch of the fissure of Sylvius running into it. This fissure of Sylvius contains in its depths a series of inturned convolutions, which are known collectively as the island of Reil. Below this fissure of Sylvius, as has already been said, is the temporal lobe, and we see in Fig. 14 the first and second temporal fissures, and the first temporal convolution lying between the fissure of Sylvius and the first temporal fissure, the second temporal convolution between the first and second temporal fissures, and the third temporal convolution below the second temporal fissure. Back of the ascending parietal convolution will be noticed a long curving fissure known as the parietal or inter-parietal fissure. This serves to divide the parietal lobe irregularly into two lobules, the upper and the lower. These upper convolutions of the frontal and parietal lobes will be better seen in Fig. 15. The convolution curving around the upper end of the fissure of Sylvius is known as the supra-marginal convolution, and the one around the upper end of the first temporal fissure is the

Fig. 16.

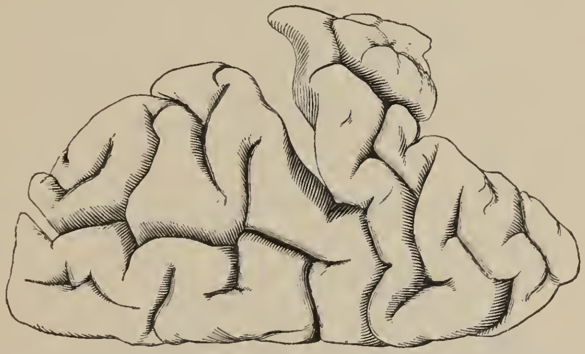


Vertical section through the centre of the corpus callosum, showing the convolutions of the inturned surfaces of the hemispheres.

angular convolution, or gyrus angularis. It will be observed that the supra-marginal convolution and the angular convolution both really belong to the lower parietal convolution or lobule. The boundary line between the parietal and occipital lobes is at the parieto-occipital fissure, which is a little indentation at the point indicated in Fig. 14, and better seen upon the inturned surface of the hemisphere (as in Fig. 16) than upon the outward lateral surface. The convolutions of the occipital lobe are illy defined upon the external hemisphere, but they can usually, with some approach to accuracy, be marked off into the first, second, and third occipital convolutions, as indicated in Figs. 14 and 15. Upon the inturned surface of the hemisphere, Fig. 16, however, there is a convolution of the occipital lobe, known

as the cuneus, from its wedge shape, and this is the most important portion of the occipital lobe for us to know. The upper boundary of the cuneus is the parieto-occipital fissure, whilst the lower boundary is the calcarine fissure, the name being derived from the adjacent hippocampus minor, which is also called the *calcar avis* or cock's spur. Upon this inturned surface of the hemisphere are a number of other convolutions with which we must become familiar. In front of the cuneus is seen the præcuneus. In front of this again is the so-called paracentral lobule, which consists of the surface inturned upon this aspect of the brain of the two ascending convolutions, the ascending parietal and the ascending frontal. At the anterior boundary of the paracentral lobule is seen the calloso-marginal fissure curving forward, downward, and backward. Above this, in front of the paracentral lobule, is the inturned surface of the first frontal convolution, whose outer surface upon the hemisphere we have seen in Fig. 14. Curving around the corpus callosum is the gyrus forni-

FIG. 17.



Form of convolutions of the upper and lateral surface of the hemisphere.

catus or arched convolution, the longest of all the cerebral convolutions. It is sometimes called the limbic lobe. Its arch around the corpus callosum is seen in Fig. 16, being known in its anterior and middle portions as the gyrus fornicatus, but in its posterior and lower portions being continuous with the hippocampal[1] and the uncinate[2] convolutions. The rear portion of the hippocampal convolution is split by the junction of the parieto-occipital and calcarine fissures, below which junction are the lingual and the fusiform lobes, as will be seen in Fig. 16. The form of a portion of these different convolutions of the upper and lateral surface of the hemispheres can be better studied in detail in Fig. 17.

The base of the hemisphere is represented in Fig. 18. The convolutions of this surface of the brain are of no practical importance

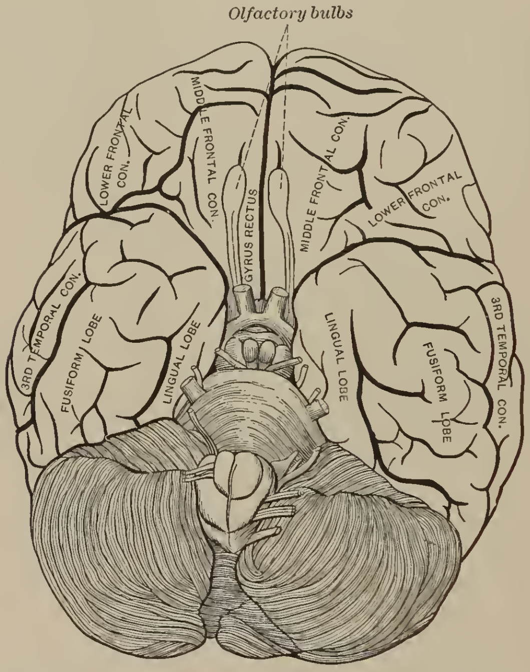
[1] The hippocampal convolution, or gyrus hippocampi, is so called because it is adjacent to the hippocampus major or the cornu ammonis, which is an eminence along the floor of the adjacent horn of the lateral ventricle.

[2] The uncinate convolution, from *uncinus*, a hook or barb, derives its name from the fact that it is of a hook shape, and is considered as belonging to the hippocampal convolution.

to us in the present state of our knowledge of localization, and they are sufficiently indicated by the lettering of Fig. 18, so I shall not pause to dwell upon them, the more especially as we shall have again to consider them when speaking of the other structures at the base.

We must now pause to state our present knowledge of the localization of functions in the different convolutions of the hemispheres. It is true that Bouillaud recognized aphasia clinically in 1825; that Dax, in 1836, located this symptom-group in the frontal lobe; that Broca more precisely located it in the third frontal convolution in 1863; that Andral, in 1834, had observed paralysis of the arm and

Fig. 18.



Convolutions of the base of the cerebrum and the cerebellum.

leg from cortical disease; that Panizza, in 1805, reported two autopsies which showed that the function of sight was somewhere in the parieto-occipital region of the hemisphere. Nevertheless, the first proof that commended itself to the minds of medical men of there being localized areas of function or so-called centres in the cortex was afforded by the world-famous experiments of Fritsch and Hitzig in 1870. In the twenty-five years which have since elapsed, not only has abundant confirmation been afforded of the statements of these two German observers, but other centres have been found in the convolutions. This has been done by the several methods that have been mentioned at the beginning of this chapter. Let us now see what we know about these cortical centres. The ascend-

ing frontal and the ascending parietal convolutions, the rear ends of the frontal convolutions immediately in front of the ascending frontal (Fig. 14), and that inturned surface of the ascending parietal and the ascending frontal convolution which is known as the paracentral lobule (Fig. 15), contain the motor centres. If the ascending frontal and the ascending parietal convolutions, with the bases of the two frontal convolutions in front of the ascending frontal, are divided approximately into four portions, the upper fourth, together with the paracentral lobule, will contain the centre for the leg of the opposite side of the body, the middle two fourths will contain the centre for the arm upon the opposite side of the body,

Fig. 19.

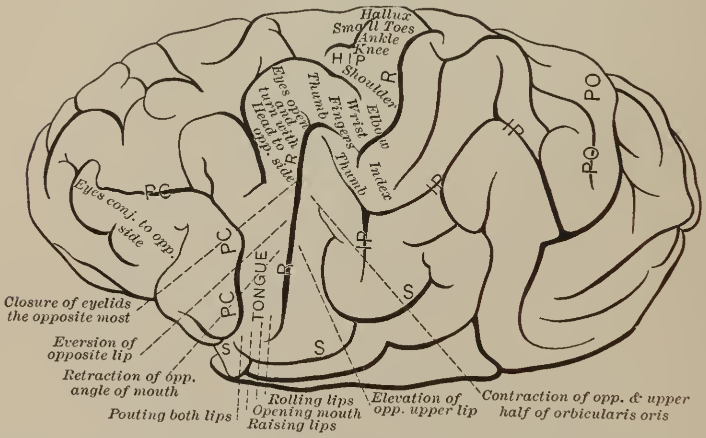
Centres of the cortex of the human brain, according to Ferrier.

1. Centre for the movements of leg and foot in locomotion. 2, 3, 4. Centres for various complex movements of arms and legs, as in climbing, swimming, etc. 5. Extension forward of arm and hand. 6. Supination of hand and flexion of forearm. 7, 8. Elevators and depressors of the angle of the mouth, respectively. 9, 10. Movements of lips and tongue as in articulation. 11. Centre of platysma retraction of the mouth. 12. Lateral movements of the head and eyes, with elevation of the eyelids and dilatation of the pupils. *a, b, c, d.* Centres of movement of fingers and wrist.

and the lower fourth (together with the portion of the third frontal fissure lying posterior to the ascending branch of the fissure of Sylvius, the so-called *operculum*) will contain the centre for the muscles of the face, the tongue, and probably also the larynx. It must be remembered that this division into fourths is purely arbitrary, and that it is utterly impossible to say just where one centre commences and where another terminates; indeed, as we shall have occasion to see, the centres of the cortex shade into one another to a certain extent. There have been considerable discussion and consequent variance of opinion among physiologists and pathologists as to the subdivision in these fourths of the motor area. Thus Ferrier minutely subdivides this motor region, as is indicated in Fig. 19,

basing his localizations largely upon his experiments on the brain of the monkey, although he is particular to state that the centres of the human brain, as he has indicated them by this method of analogy, are only approximately correct, inasmuch as the movements of the hand and arm, for instance, are more complex and differentiated in man than in the monkey, whilst in the human being there is nothing to correspond with the prehensile movements of the lower limbs and tail of the monkey. Horsley and Beevor have outlined this region in a somewhat similar manner to Ferrier, also by experiments upon animals. (Fig. 20.) The third frontal convolution (Fig. 14) is the centre of speech in the sense that a lesion of it causes an inability to articulate words properly, although the patient may understand perfectly well what is said to him. This is called *motor aphasia*, and was the symptom to which Bouillaud, Dax, and Broca first called

FIG. 20.



Outer surface of the left half of the brain of an orang-outang, showing centres. (HORSLEY.)
R. Fissure of Rolando. IP. Interparietal fissure. PO. Occipito-parietal fissure.
S. Fissure of Sylvius. PC. Præcentral fissure.

attention. Lesion of the first temporal convolution, and to some extent of the second temporal convolution, especially, as Mills has recently shown, in their upper and posterior portions, gives rise to the symptom which is known as *mental* or *word-deafness*, characterized by the ability of the patient to hear perfectly well what is said to him, at the same time that he fails to understand what he thus hears. Singular to say, the most beautiful exposition of this was given by Munk through his experiments upon the brains of dogs. He would so train a dog as to have him crouch at the word of command or at the crack of the whip. From this dog's brain he then removed the auditory centre in the convolution analogous to the temporal convolution of the human being, then stuffing the ear upon the side of the uninjured nerve, so that the dog could hear nothing in that way. Coming into the room suddenly behind the animal, and speaking to him or cracking his whip, the dog would evidently hear

what was said to him, as well as the crack of the whip, because he would prick up his ears and manifest other signs of audition, but he would not crouch, as he had been accustomed to do, showing that although he heard the sounds, he no longer understood. If the cotton was now taken from the uninjured ear, he again not only heard the sound, but crouched at the word of command or the crack of the whip—thus evidencing that, through the sound side of the brain, he not only heard but understood. When there is a lesion of the island of Reil, which, as has been said, is a series of small convolutions buried in the fissure of Sylvius, there is caused what is known as *paraphasia*, or the *aphasia of conduction*. In such a case patients understand perfectly well what is said to them, and articulate words correctly, but they misplace them, saying, as did one patient of mine, "I see best," instead of "I have been;" and making the most curious and insensate combinations of words. A patient suffering from paraphasia may be able to speak a given word himself, but cannot repeat it when it is spoken by another. A lesion in the angular gyrus generally causes what is known as *mental* or *word-blindness;* that is, an inability to recognize the meaning of a word which is seen, although it may be automatically repeated. This word-blindness is usually associated with true blindness of that variety known as hemianopsia, which is described further on. Word-blindness and word-deafness are frequently observed together in the same patient, due to consentaneous implication of the temporal and the angular convolutions, as might be expected from the proximity of the posterior end of the temporal convolutions to the angular convolution. There is a symptom known as *agraphia*, consisting of an inability to write, although there may be no paralysis of the hand, no loss of the memory of words, no word-blindness, and no hemianopsia. It is by no means certain as to where its causative lesion is situated, although possibly, as has been supposed, it may be in the posterior portion of the second frontal convolution. The cuneus, a small portion of the adjacent occipital lobes, and the lingual lobule, constitute the centre of vision (Fig. 16) in the sense that a lesion in this region causes blindness of the lateral half of the eye to the opposite side of the median line—*i. e.*, if the right occipital lobe in this region is affected, the patient fails to see anything to the left side of the middle line of the eye. This defect of vision is that which is known as hemianopsia.[1] There is a considerable doubt at the present day as to exactly where the centres are of the sensations of touch, pain, muscular sense, and temperature. Ferrier and his followers maintain that the site of the tactile sense is in

[1] Hemiopia is derived from the Greek words ἡμισυς, "half," and ὄψις, "sight," meaning, therefore, half sight. There is another somewhat similar word, which has led to much confusion—hemianopsia, from ἡμισυς, "half," ἀν (privative) and ὄψις, "sight," meaning also half sight. It seems to be agreed at the present day that by arbitrary custom hemiopia shall mean the condition of the retina, while hemianopsia shall be applied to the crossing of the rays of light in the media in front of the retina. Thus a left *hemiopia* will indicate that the left halves of both retinæ are blind, so that the patient, not being able to see objects to the right of either eye, shall be said to have a right *hemianopsia*. This is made plain by the diagram of Fig. 28.

the gyrus hippocampi (Fig. 16), and Schäfer and Horsley formerly believed that it was in what Broca has termed the limbic lobe, by which, as has already been explained, he meant the gyrus fornicatus as it sweeps around the corpus callosum, ending below in the hippocampal and uncinate convolutions (Fig. 16). Others have shown, however, that lesions of the motor area may have with them some

Fig. 21.

Localization of muscular sense, with motor paralysis, in a case of my own, with autopsy.

impairment of sensation. I have myself recently reported a case in which a round-cell sarcoma was found in the ascending parietal convolution at about the junction of the upper and middle third, from which there had resulted a motor paralysis of the opposite arm and leg with a distinct impairment of muscular sense (Fig. 21). Horsley, in a recent article, stated his belief that there is impairment of muscular sense in the lesions of the motor area, and reported a case of removal of a tumor from the centre for the thumb (Fig. 20), in which, during recovery, there was considerable pain in this member.

Fig. 22.

Diagram of lesion causing loss of tact and pain sense, with preservation of muscular sense (autopsy).

I have recently seen a case in consultation in which there was absolute loss of the sense of touch and pain, with preservation of the muscular sense, and in which the autopsy, made by my friend Dr. Frank Madden, of Plattsburg, New York, disclosed a lesion of the parietal lobe, as represented in Fig. 22. In the last few years there has grown to be a belief that the parietal lobes are the terminals of

PLATE I.

FIG. 1.

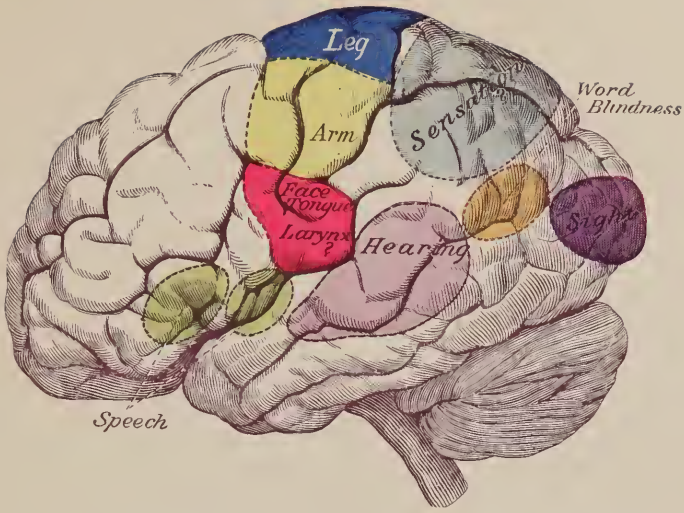


CHART OF LOCALIZATION OF CORTICAL CENTRES DETERMINED ON EXTERNAL SURFACE OF CEREBRUM.

FIG. 2.

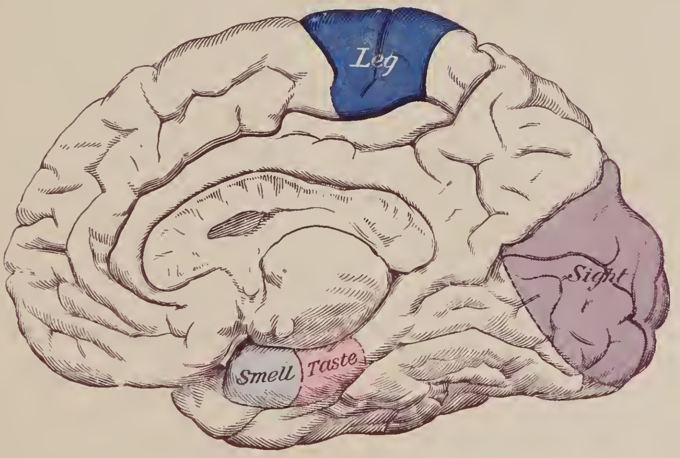


CHART OF LOCALIZATION OF CORTICAL CENTRES DETERMINED ON MEDIAL SURFACE OF CEREBRUM.

[To face page 42.

sensory nerve-fibres of the general senses of pain, muscular sense, tact, and perhaps heat; but the proof of this has been rather anatomical than physiological. There might, indeed, be a question as to whether it is necessary to suppose that there are centres of sensation distinct from those of motion, if we accept the views of Cajal and Andriezen, that are soon to be explained, of the mode of entrance of the sensory nervous current to the motor cells of the cortex.

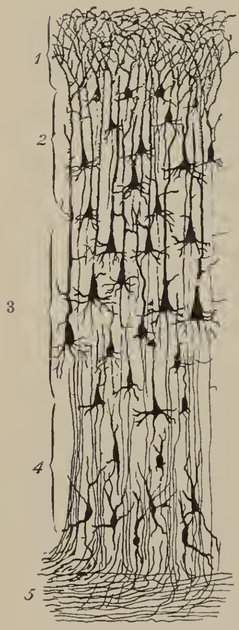
It will be thus seen that our knowledge of the centres of the cortex is remarkable when we consider that almost the whole work of discovery and confirmation has been done since 1870. Plate I. represents the centres that are known as we have been describing them. From this it will be seen that we know the motor centres for the arm and leg muscles, for the muscles of the head, face, and larynx, for articulate speech, for word-deafness, for paraphasia or interruption of speech-conduction, for word-blindness, for hemianopsia, and possibly for agraphia; whilst the centres for the muscular sense, for tact, and for pain are possibly to be found in the parietal lobes. There is some dispute, however, as to the exact localization of the different muscular groups in the motor region. It is very evident that there is a large portion of the cortex with whose functions we are not acquainted. The portion of the two frontal convolutions somewhat in front of the ascending frontal convolutions are inexcitable, and lesions of them do not give rise to motor or sensory phenomena, but rather to purely mental phenomena, so far as we now know. Outside of the regions we have marked, the functions of the cerebrum are unknown to us (Plate I.). It is probable that every year will add to our knowledge. The doctrine of cerebral localization has been vigorously opposed by a small and constantly waning school, of which Goltz is the most illustrious exponent. As critics these writers have been of great service to science, for they have exposed the absurd claims of over-enthusiastic localizationists, and have shown that, in order to destroy or even distinctly impair a centre of the cerebral cortex, a lesion must be destructive, and not merely irritative; thus, whilst a tumor, a softening, or a marked compression from a fracture may inhibit or destroy the function of a centre, this may persist with a mild degree of meningitis, especially of the pia mater, or with slight compression. The more localized a lesion is, too, the more prone it is to affect the centre. But Goltz and his followers have not succeeded in their attempt to demonstrate that the doctrine of cerebral localization is based on faulty observation—indeed, this is the very defect of all their own reports of cases or experiments. Speaking from my own clinical experience, I can say deliberately that I have never seen a case disproving cerebral localization, although I have observed and read of many in which an imperfect history made it impossible to reach a conclusion.

The histology of the gray matter of the brain is quite intricate and differs somewhat in different regions. The late researches, to which I have so frequently alluded, have made considerable modifications even in the views that were current at the time of the first edition of

this book, two years ago. Let us, for example, examine the type of structure that is seen in the motor area. This has been described as consisting of five layers, and even of six, but the present opinion is that there are only four, viz., the molecular layer, that of the small pyramidal cells, that of the large pyramidal cells, and that of the polymorphous cells.

The molecular layer (1, Fig. 23) presents a finely granular or reticulated appearance. It is dotted here and there with some nuclei probably belonging to the neuroglia cells, especially abundant beneath the pia mater, and it also contains a few other and larger nuclei, surrounded by a triangular or fusiform protoplasmic body that is probably of nervous function. In the most superficial portion of this molecular layer Köllicker and Exner discovered a group of horizontal fibres. Andriezen has recently described in the outermost region of this molecular layer a system of neuroglia cells, giving rise to a tangential plexus of fibres radiating from the bases of these cells and also to tufts of descending fibres. This molecular layer, however, is worthy of more extended study, because of the fact that the earliest lesions of general paresis are probably found in it, whilst it is probably affected in other mental affections. Fig. 24 shows some late remarkable researches regarding it by Cajal. It would appear that its special cells are astonishingly peculiar in the unlikeness of their processes to the usual protoplasmic and axis-cylinder branches of other nerve-cells. There are three varieties of these nervous structures, namely, polygonal, fusiform, and triangular or star-shaped cells. The polygonal are of medium size (Fig. 24, *D*), and have four to six protoplasmic processes, which are nodular, fine, ramifying, and divergent, some of them descending to the layer of the small pyramidal cells. The axis-cylinder process is fine, proceeding from the side of the cell or of a large process, and it passes obliquely or horizontally into the thickness of the molecular layer, ramifying greatly and breaking up into a certain number of wavy terminations which are very long and parallel to the surface of the cortex, but never descending to the white matter as do the axis-cylinders of the pyramidal cells. The fusiform (*A*, *C*) and the triangular (*B*) are cells special to this molecular layer in that their axis-cylinder processes cannot be distinguished from the protoplasmic ones. Another striking peculiarity of these two cellular types is that they possess not only one, but two or more nervous

Fig. 23.

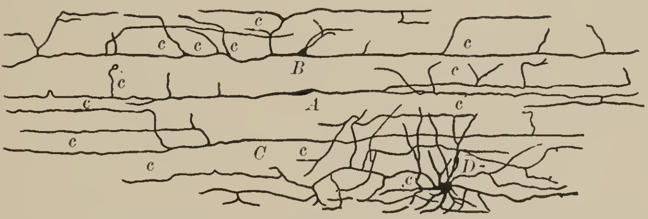


Perpendicular section of the gray matter of a cerebral convolution. (Cajal.)

1. Molecular layer; 2. Layer of the small pyramidal cells; 3. Layer of the large pyramidal cells; 4. Layer of the polymorphous cells; 5. White matter. (Cajal.)

processes extending horizontally in the thickness of the molecular layer, where they divide frequently at a right-angle, thus traversing an enormous space. The fusiform cell is ovoid (*A* and *C*), lying along a line horizontal with the surface of the cortex, and possessing two large polar trunks which are almost rectilinear. These trunks, as has already been said, give off numerous branches at a right angle, then curve so as to approach the surface of the cortex, and finally break up into two, three, or more filaments which are very long and wavy, and which ramify exclusively, almost always at a right-angle, in the molecular layer. During their course, often at the level of the bend (Fig. 24, *C*), these polar trunks emit very fine horizontal branches which are also very long, and which themselves are provided with ascending collaterals. Some of them, as at *A*, Fig. 24, however, do not change their direction, but gradually dwindle in size until they seem to become a simple or double nervous filament. The triangular or star-shaped cell (*B*) is different from the fusiform one in that the cell-body possesses three or four polar

FIG. 24.



Special cells of the molecular layer of the cerebral cortex of a dog one day old. (CAJAL.)
A. Fusiform cells; *B*. Triangular, or star-shaped cell; *C*. Another fusiform cell; *D*. Polygonal cell with numerous protoplasmic processes, and one axis-cylinder divided several times: *c*. Axis-cylinder.

branches which take a horizontal direction through a long tract, and gradually dwindle into a filament like that of an axis-cylinder, and during their course they give off, like the fusiform cell, a number of fine collaterals which cannot be distinguished from other nervous filaments of the molecular layer. According to Cajal, these fusiform and star-shaped cells are found in the molecular layer in all regions of the cortex, most especially in the deeper or internal half of the former; whilst the ordinary star shaped cell, with only one axis-cylinder, is generally in the external half. To the nervous filaments of the molecular layer thus made by the axis-cylinders and these peculiar cells, there are added a number of nerve-fibres coming from the pyramidal cells, as we shall soon have occasion to see.

The layer of small pyramidal cells is composed of polyhedral or pyramidal cells (2, Fig. 23) of moderate size, their dimension and the length of their trunk increasing from above downward. This trunk, which is often split into two near the body of the cell, terminates in a large plume or *bouquet*, and occupies, interlacing with plumes from neighboring cells, almost the whole thickness of the

molecular layer. The axis-cylinder is very fine, descends, and splits up into four or five fine collaterals, each of which in its turn divides into two. Golgi supposed that the collaterals of these axis-cylinders anastomosed with each other to form a continuous network in the gray matter. Cajal, however, has traced them in a thoroughly satisfactory and at the same time extremely interesting manner. Finding that they were extremely long in the human being and the large animal, he searched for them in the embryo and in the newly born small mammals, and ascertained that they terminated with a varicose expansion without any interlacing; and he has even followed them in the young fœtus from their very outgrowth from the axis-cylinder up to their final dichotomous division. The layer of small pyramidal cells merges insensibly with the large one of the succeeding cells.

The large cells of the pyramidal layer (3, Fig. 23) have a conical or pyramidal shape, with an inferior base from which issues the axis-cylinder. The protoplasmic processes are numerous and are divisible into the ascending trunk or primordial expansion, the collaterals of the ascending trunk, and the basilar expansions or processes of the cellular body. The ascending trunk is thick and points to the surface of the cortex parallel to the trunks of other pyramidal cells. When it has arrived at the molecular layer it expands into a plume or *bouquet* of protoplasmic branchlets, which terminate free among the nervous filaments of this zone, and the commingling of all these plumes or bouquets gives rise to a very complicated protoplasmic plexus, to which is due the finely reticulated appearance of this portion of the cortex in the ordinary carmine staining. Golgi supposed that these protoplasmic branchlets were in connection with the vessels and the neurogliar cells, but Cajal positivelystates that they distribute themselves throughout the whole thickness of the molecular layer, and Retzius has verified this. The axis-cylinder of these pyramidal cells passes through all the lower layers of the cortex and into the white substance, where it bends and becomes continuous with a nerve-tube. During its course it gives off six to ten fine collaterals at a right-angle, which proceed horizontally or obliquely, and split up into two or three terminal branches. Cajal calls these pyramidal cells the *psychical cells.* They increase in their complexity the higher the scale of creation rises, being exceedingly simple in the frog and the lizard, beginning to be complicated in the rat, and reaching their highest evolution in man.

The layer of the polymorphous cells contains a few pyramidal cells (4, Fig. 23), some of which are large, and some of medium size, whose trunk is directed toward the molecular layer. But the most of the cells are ovoid, fusiform, triangular, or polygonal. There are certain characteristics of almost all the cells of this layer. The trunk is usually of less size and expansion, so that it never reaches the molecular layer or the terminal *bouquets* of the pyramidal cells. In some of them there is no trunk, its place being taken by two or more short and oblique processes, and there are occasionally others with three protoplasmic expansions, of which two pass to the white substance. The axis-cylinder furnishes two to four collaterals which

ramify several times and pass by a bend or a T-shaped division into one or two nerves of the white substance.

Intermingled in small number with the elements of the molecular and the small and large pyramidal layers are two species of cells known as the sensitive corpuscles of Golgi, and the cells of Martinotti, with ascending axis-cylinder. The former are large and polygonal and have protoplasmic expansions all around the body, whilst the axis-cylinder comes off from the upper or lateral part of the body, marches in various directions, and after a short distance splits up into a free arborization surrounding the body of the neighboring cells. Golgi supposed that the shortness of the axis-cylinder was an evidence of the sensory function of the cell; but Cajal suggests that these short axis-cylinders are probably only meant to connect with neighboring cells, and this seems much more reasonable, because we have no ground for supposing that a sensory axis-cylinder needs to be any shorter than a motor one, inasmuch as it carries its messages through equal lengths of tissue. The cells of Martinotti, with an ascending axis-cylinder, are especially to be found in the layer of the polymorphous corpuscles, although, as has been said, they are found in some degree through all the first three layers. They are fusiform or triangular, with protoplasmic expansions that are either ascending or descending, but the axis-cylinder, which may be emitted from an ascending protoplasmic trunk, mounts almost like a straight line to the molecular layer, where it divides into two or three large branches ramifying horizontally and splitting up into a terminal arborization of great amplitude. With some cells it has appeared to Cajal that this terminal arborization was among the small pyramidal cells.

Fig. 25.

Section magnified 3.75 diameters, showing the nerve-fibre passing into a convolution.

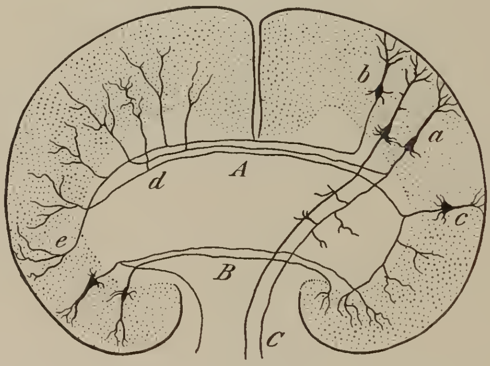
Into these layers of the brain-cells ascend the medullated nerve-fibres from the sublying nervous regions, as is represented in Fig. 25, which is drawn from a microscopical section of a convolution, and in which the nerve-fibres are seen coming up in a sheath or bundle and diverging gracefully to penetrate the gray matter. The medul-

lated nerve-fibres constitute the white matter of the brain, and are of four varieties:

1. Projection-fibres;
2. Commissural fibres;
3. Association-fibres;
4. Centripetal or terminal fibres.

The projection-fibres (*C*, Fig. 26) come from all the regions of the cortex and converge toward the corpus striatum to pass into the crura cerebri. In the small mammals they emit, at the height of the corpus callosum, a large collateral for this body, and then they descend in small tracts separated by layers of gray substance, which they furnish with very fine collaterals, although there are some of them which maintain their individuality, without giving off a single collateral throughout the whole depth of the corpus striatum. Molokoff and other authors supposed that these projection-fibres were the sole continuations of the large pyramidal cells, whilst the association and the commissural fibres came from the small pyramidal cells. Cajal, however—although he is careful to explain that his proof is not as yet satisfactory—maintains that they come from both the large

FIG. 26.



Transverse section of the cerebrum, showing the probable disposition of the commissural and projection-fibres. (CAJAL.)

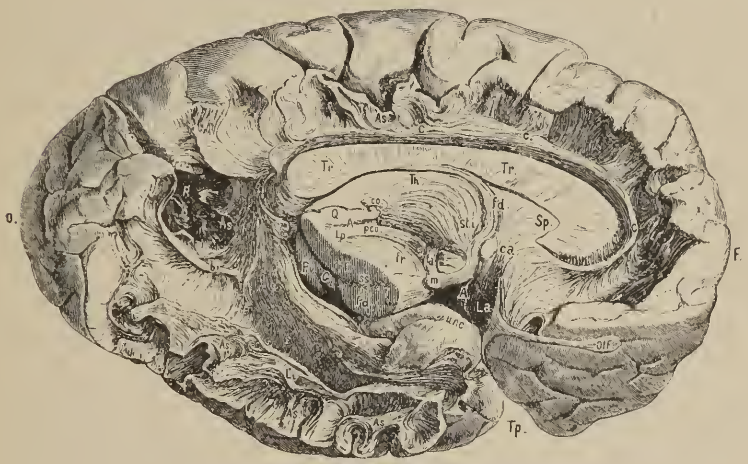
A. Corpus callosum; *B*. Anterior commissure; *C*. Pyramidal pathway formed of the projection-fibres.

and the small pyramidal cells, and even from certain of the polymorphous cells, and he suggests that this origin from cells of different dimensions explains why the axis-cylinders are of varying size. There is every reason to believe that their lower passage is through the pyramidal fibres of the spinal cord, although the proof of this rests upon the well-known pathological methods, and has received as yet no confirmation or negation from the latest histological investigators.

The association-fibres are divisible into short and long ones; the short ones, the so-called proper arcuate fibres, connect each two neighboring convolutions of the same hemisphere. Besides these, the tangential fibres really belong to this system (Fig. 29). The

long fibres of association furnish a connection between the more distant convolutions of the hemisphere, and they consist of the superior longitudinal fasciculus, whose fibres run in the direction of the second frontal convolution, and connect the frontal convolutions with the occipital convolutions; of the sub-callosal longitudinal fasciculus, running under the corpus callosum and also connecting the frontal lobes with the occipital; the inferior longitudinal fasciculus, running to the side of the posterior horn of the lateral ventricle and connecting the occipital lobes with the temporal; the uncinate fasciculus, crossing the entrance of the fissure of Sylvius and connecting the lower frontal convolution with the tip of the temporal lobe; the cingulum, passing from the anterior perforated space through the gyrus fornicatus to the tip of the cornu ammonis, its anterior portion connecting the olfactory bulb with the frontal lobe, and its posterior part connecting the hippocampal gyrus with the temporal lobe; the

FIG. 27.



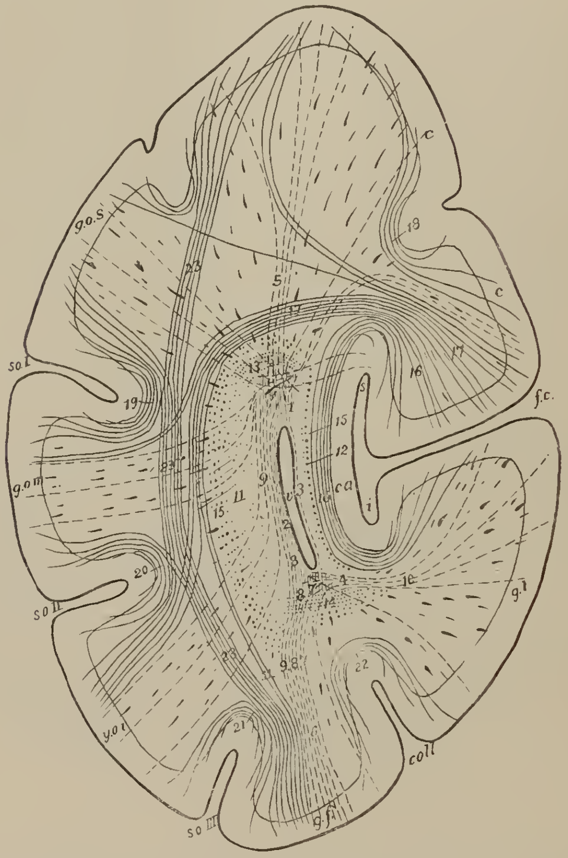
Tearing of the cortex and the subcortical white matter along the medial surface of the cerebrum. (From MEYNERT.)

F., Tp., O. Frontal, temporal, and occipital regions. Tr. Corpus callosum. C. C. Cingulum, surrounding the corpus callosum and beneath the calloso-marginal fissure. As. Association fibres, *i. e.*, fibres connecting adjacent convolutions. R. Cortex. bi. Internal basal fasciculus of Burdach. Li. Inferior longitudinal fasciculus. Olf. Olfactory lobe. La. Lamina perforata anterior. ca. Anterior commissure. unc. Hook of the hook-shaped convolution of gyrus uncinatus. Sp. Septum pellucidum. Th. Optic thalamus. f.d. Descending fornix. m. Corpus medullare. f.a. Ascending fornix. Q. Corpora quadrigemina. A. Aqueduct of Sylvius. Pv. Pulvinar of the optic thalamus. Gi. Internal geniculate body. T. Tegmentum. Pd. Pes of crura cerebri. St i. Internal stylus of the optic thalamus. Lp. Posterior longitudinal fasciculus. co. Conarium.

vertical fasciculus, which brings the lower parietal lobe into connection with the fusiform lobule. (Fig. 27.) In the cerebellum there are also association-fibres passing from convolution to convolution, the so-called garland-shaped fibres of Stilling. Those of the cerebrum probably arise from the three layers of cortical cells—*i. e.*, the large and small pyramidal and the polymorphous cells; but as yet all

that has actually been observed has been their connection with the polymorphous cells, and occasionally with a large pyramidal cell. The axis-cylinders of these cells connect with the association-

FIG. 28.



Frontal section of the occipital lobe showing the association-fibres, according to SACHS, of Breslau.

v. Descending cornu or horn of lateral ventricle. *f.c.* Calcarine fissure. *s.* Upper cleft of calcarine fissure. *i.* Lower cleft of calcarine fissure. *coll.* Collateral sulcus. *s.o.I.* Parietal fissure. *s.o.II.* First occipital fissure. *s.o.III.* Second occipital fissure. *c.a.* Calcar avis or hippocampus minor. *g.l.* Lingual lobe. *g.f.* Fusiform lobe. *g.o.s.* First occipital convolution. *g.o.m.* Second occipital convolution. *y.o.i.* Third occipital convolution. *c.* Cuneus.

The following are the different strands which Sachs has marked out:

1–10. The forceps. 11–14. Internal sagittal stratum. 15. External sagittal stratum. 16. Calcarine stratum. 17. Transverse stratum of cuneus. 18. Stratum proprium of cuneus. 19. Stratum proprium of parietal fissure. 20. Stratum proprium of first occipital fissure. 21. Stratum proprium of second occipital fissure. 22. Stratum proprium of the collateral sulcus. 23. Deep stratum of convexity.

fibre usually by a simple bend, but occasionally by a T-shaped division into equal or unequal fibres; so that many of these association-fibres, by means of the direction of the different connections of the

two bifurcating branches, may bring a cell at one place in the cortex into relation with a large number of others in far distant regions. These association-fibres increase proportionately to the mass of the gray matter, so that in man and the large mammals they form the principal mass of the white substance. Their number and their extraordinary length, conjoined with their intricate interlacing with the projection and commissural fibres, renders it impossible to follow any one of them anatomically, and hence it is that they have hitherto only been studied by the so-called tearing method in brains that have been peculiarly hardened. But Cajal has made the happy discovery that they can be investigated with far greater readiness in the small brains of the rat, the bat, the mouse, etc., not only because of the much more minute dimensions, but also because, singular to say, in certain places the association, commissural, and projection-fibres are perfectly delimited. Cajal has also demonstrated that in many of the association-fibres there are very often collaterals which ascend and ramify in the different layers of the cortex, some of them going as far up as the molecular layer, and others as far down as the white substance. These association-fibres terminate, like the fibres in the spinal cord, by means of free, wavy arborizations extending throughout the whole cortex, even to the molecular layer.

The commissural fibres are to be found in the corpus callosum (*A*, Fig. 26) and the anterior commissure (*B*). The former consists mainly of fibres running from one hemisphere to another, passing horizontally across and then bending so as to diverge fan-like into different but always identical portions of the hemispheres, with the exception of the temporal and the basal portions of the occipital lobes and the olfactory bulbs. The anterior commissure of the same side is an addition to the corpus callosum, and connects those identical portions of the hemispheres which the corpus callosum does not join. In the cerebellum there are also commissural fibres connecting identical portions of the two hemispheres, and again in the brain-stem between the identical nuclei of the cranial nerves, and still again in the spinal cord, in which the fibres of the posterior commissure connect the cells of the two posterior horns. The commissural fibres (*A* and *B*, Fig. 26) are situated beneath those of association and maintain a transverse direction in the small mammals. They are extremely minute, and Cajal has determined that they have a medullary sheath. They arise in different regions of the cerebral cortex of one hemisphere and terminate in the other, but in the sphenoidal lobes they are aggregated into a separate tract to form the anterior commissure. Many of them give off very fine collaterals at a right angle, generally to the number of two or three, and these ascend to be lost in the gray matter above, where they terminate in a free end. Some of them even bifurcate, one of the divisions passing horizontally and the other penetrating the gray matter (*c*, Fig. 26). It would seem probable that these commissural fibres are made up of direct axis-cylinders and of collateral fibres of other axis-cylinders, because, on the one hand, certain of them have been traced as collaterals of either association- or projection-fibres, whilst, on the other hand, other fibres

have been followed from different layers of the cortex, principally from the level of the small pyramidal cells, down to the corpus callosum, into which they pass with a bend. This, however, it should be borne in mind, is not yet positively determined. The termination of the commissural fibres is still more uncertain, although some of them have been seen ascending through the gray substance and there ramifying, but as yet their terminal arborization has not been actually observed. Sufficient, however, is known of them to enable us to regard them as nervous connections between symmetrical regions of the same hemisphere, as well as probably also between similar elements of the different regions and layers of the cortex.

We have learned much in the last few years of the nervous connections between the different layers of cortical cells. Certain thick fibres have been observed passing obliquely or horizontally through the gray substance and ramifying, throughout the whole thickness of this region, even the molecular layer, with a ramification of enormous extent (Fig. 30, E). These are supposed to come from the spinal cord, the cerebellum, etc. The terminal branches of these ramifications are varicose, and especially surround the small pyramidal cells. Cajal ventures the suggestion that these are probably sensitive nerves, or at least axis-cylinders coming from cells which are connected with the terminal fibres of sensitive nerves. He has observed them especially in reptiles. Be this as it may, certain facts are assured about the connection of nerve-fibres with these cells of the cortex. Fig. 29 delineates a section of the cortex stained with Weigert's hæmatoxylin upon the left side, and according to Golgi's method upon the right, showing the cells therefore on the left hand and the fibres on the right. The layers of the cells have already been explained in Fig. 23. It will be seen that the uppermost region of the cortex contains a number of fine fibres which mostly maintain a tangential direction. Beneath these tangential fibres are seen the super-radiating fibres. At a lower level still is the layer of Gennari, and at a still lower level the inter-radiating fibres, whilst lowermost of all are the radii themselves. The relation of these to the different layers and cells is easily seen from this figure. Cajal has endeavored to show the course of the nervous currents into and out of these cortical cells in the following manner (see Fig. 30): Starting from the fact that all the pyramidal cells, as has already been described, have a protoplasmic plume or *bouquet* extending into the molecular layer, he regards these structures as the receptacles of nervous currents from other cells through the axis-cylinders coming into these protoplasmic expansions. In other words, to use Cajal's terms, the current is *cellulifugal* in the axis-cylinder and *cellulipetal* in the cell-body or the protoplasmic processes, the latter receiving the currents and the former transmitting them. (See Fig. 30.) Reasoning from these premises, Cajal suggests that in the molecular layer of the cortex the plume of the pyramidal cells receives currents (1) from the special cells of the molecular layer through their nervous expansions; (2) from the vertical fusiform cells of the cortex, by means

of their upper arborizations and their ascending axis-cylinders; (3) from the pyramidal cells of association in variously distant regions of the cortex, through the medium of their ascending collaterals and the terminal arborizations of their axis-cylinders; (4) possibly also from

FIG. 29.

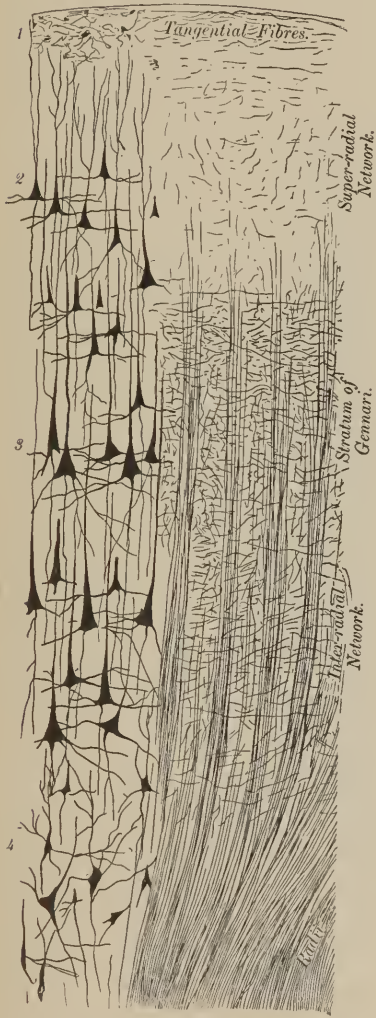


FIG. 30.

FIG. 29.—Human cortex stained with Weigert's hæmatoxylin on the left, and by Golgi's method on the right.

FIG. 30.—Probable direction of the currents and the nervous protoplasmic connections in the cell of the cerebral cortex. (CAJAL.)

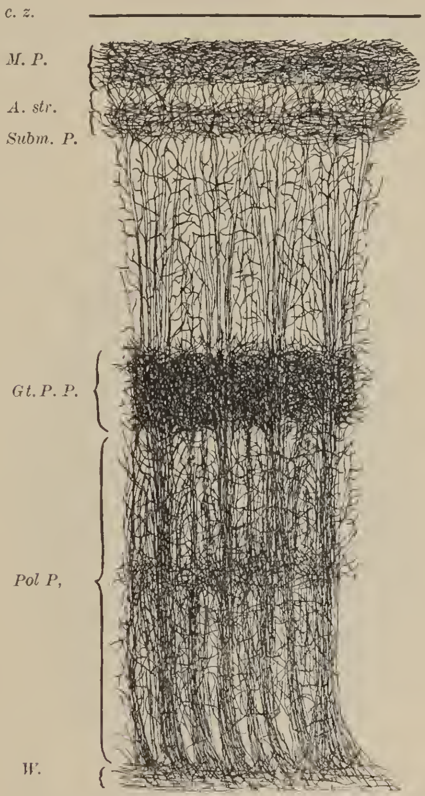
A. Small pyramidal cell; *B.* Large pyramidal cell; *C, D.* Polymorphous cells; *E.* Terminal fibre coming from other nerve-centres, *F.* Collaterals of the white matter; *G.* Axis-cylinder bifurcating in the white matter.

the cells of the cerebellum, of the spinal cord, etc., through the extended nervous arborizations which form, throughout the whole gray matter, certain large fibres coming from the white substance; (5) possibly also from the cells of the opposite hemisphere through the terminal branches of the commissural fibres. This suggestion is in the line of very rigid reasoning from the facts that have already been

explained, and it certainly offers a superb explanation, together with what follows, of the widespread commingled cerebral and physical symptoms of general paresis, in which, as we shall have occasion to see, the lesions are especially in the molecular layer. The nervous connections between the pyramidal and the polymorphous cells are extremely complicated. They are between the cellular body, the trunk, and the protoplasmic processes of these cells, upon the one hand, and five varieties of nervous fibres upon the other, which latter are constituted by the collaterals of the white substance, those of the corpus callosum, the terminal association-fibres, the terminal arborizations of the so-called sensory cells of Golgi, and the myriad collaterals coming from the axis-cylinders of the cells in the three deeper layers of the cortex. If these conjectures of Cajal's be correct, the pyramidal cells would receive in this manner nervous influences from the sensory cells of Golgi that are scattered throughout the layer of the pyramidal cells; from the cells of the association-fibres situated in the different lobules of the same hemisphere; from the same cells of the opposite hemisphere, through the medium of the commissural fibres or the anterior commissure; from the cells of the sensory areas of the cerebrum, and from the cells in the layers beneath the pyramidal cells. The last of these connections appears to be established by means of the collaterals of the axis-cylinders coming from the upper pyramidal cells, as well as by the medium of the body, the protoplasmic trunk, and the basilar processes of the pyramidal cells beneath. Each collateral is of great length, has many ramifications, and maintains a horizontal course, so that it touches the protoplasmic expansions and the bodies of many cells, enabling the small pyramidal cells to send a nervous current to a whole series of other pyramidal cells, both medium-sized and small, lying at a deeper level. Each large pyramidal cell, in its turn, owing to its large surface of contact through its trunk, its protoplasmic collaterals, and its descending basilar processes, can receive nervous currents from an infinity of pyramidal cells above it. This is represented in Fig. 30 (*A*, *B*, *C*, *D*), in which the arrows show the direction of the nervous currents. Although this matter is to some extent conjectural, yet certain fibres have been seen terminating by arborizations in the cortical layer of the optic lobe in birds, which is the equivalent of the molecular layer in the human being; in certain mammals fibres coming from the olfactory lobe send collateral and terminal branches to a superficial zone which is the place of union between the protoplasmic plumes of the pyramidal cells, and which is therefore the exact analogue of the molecular layer of the human cortex; whilst in reptiles not only the olfactory fibres, but even the other axis-cylinders, probably contain sensory fibres from the cord, and ramify in great numbers in the superficial or molecular layer of the cortex. For these reasons Cajal believes that a voluntary movement begins in the protoplasmic plume or *bouquet* of the pyramidal cells, and that therefore it has its origin in the midst of the molecular layer, and he would thus explain the fact that mechanical, chemical, and electrical stimuli of the cerebral cortex will excite movements in determinate muscular

groups. Andriezen has recently shown that the whole cortex is pervaded by several strata of nerve-plexuses, extending in sheet-like fashion. These are illustrated in Fig. 31. Each of these plexuses is for purposes of association, and contains association, commissural, and projection-fibres, which, Andriezen maintains, connect with the apical or basilar protoplasmic expansions of cortical nerve cells. Andriezen furthermore describes certain cells to which he gives the name of "ambiguous." These are contained in the molecular layer, and

Fig. 31.



Cortex of the human brain, showing the nerve-fibre systems and plexuses (combined Weigert's and Golgi's methods).

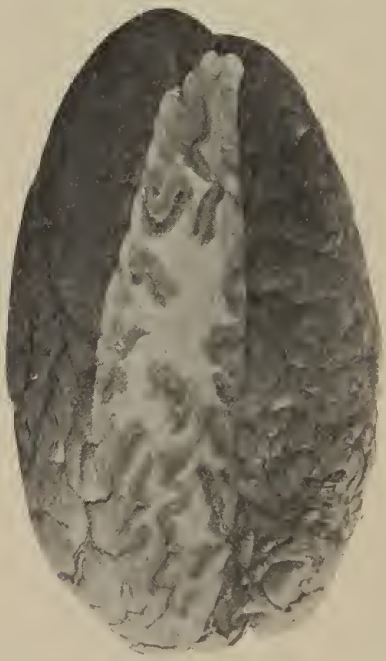
c. z. Clear zone (free from nerve-fibre). *M. P.* Molecular plexus (Exner's) in the molecular layer. *A. str.* Ambiguous cell-stratum. *Subm. P.* Submolecular plexus. *Gt. P. P.* Great pyramidal plexus. *Pol. P.* Polymorphic plexus. *W.* White substance. (Andriezen.)

they are fusiform, pyriform, triangular, or polygonal in shape, but often bifurcated into two unequal horns. He regards them as distinct in an anatomico-physiological way, and as receiving and distributing sensory excitations in the same manner as the pyramidal cells.

This cortex or gray matter of the cerebrum is the organ of the mind, and the so-called centres are simply the terminal stations of the different peripheral nerves which are thus brought into connec-

tion with the mind as a whole. The centres are therefore only compartments of the mind, and clinical observation constantly shows that lesion of any one of them need not cause mental impairment; but that, to produce disorder of the mind, there must be more or less widespread disease of the cortex involving a number of centres. On the other hand, as we shall have occasion to see when studying the insanities, mental disease may exist alone without the physical symptoms that demonstrate lesion of the individual centres. Through the influence largely of the teachings of the German school it is generally supposed that the frontal lobes anterior to the motor area must be affected in order to have mental disease; but this has been again and again disproved by clinical observation, as lesions in the different lobes of the brain have been quite sufficient to cause marked

Fig. 32.



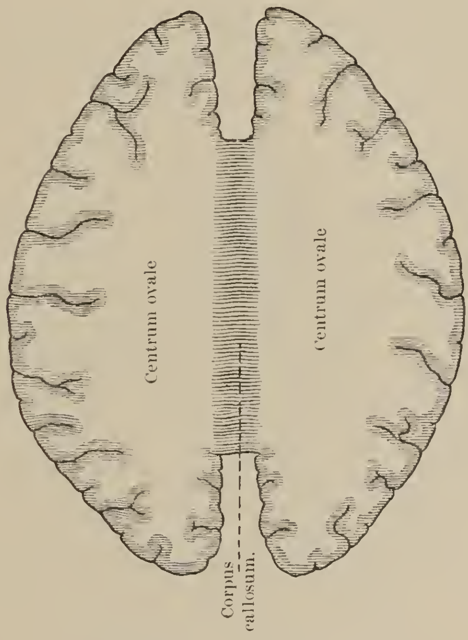
Section of the cerebrum, showing the centrum ovale on the left side.

mental symptoms. We are therefore warranted in regarding the cortex, as a whole, as the organ of the mind, and the so-called centres as being those areas of the cortex in which terminate the nerves coming from the periphery, or what are known as the projection-fibres, in contradistinction to the commissural and association-fibres of the cerebrum, which, as we have seen, connect the different convolutions.

It will now be our task to travel the highly complex route which these projection-fibres take from the cortex to the peripheral struc-

tures, although it must be borne in mind that in doing this we can no more than outline the anatomical facts which have a clinical bearing. Fig. 32 represents upon the left side a transverse section made across the hemisphere a short distance below the vertex, and shows the centrum ovale, consisting of the white matter in the centre, fringed around with the convolutions. Fig. 33 represents a still deeper section of the hemisphere, showing in the middle line the great mass of commissural or connecting fibres running across from one hemisphere to the other, the so-called corpus callosum. When this corpus callosum is lifted (Fig. 34) the ganglia of the base, or the *basal ganglia*, are seen lying in the ventricles, the third ventricle in the centre and the lateral ventricles one on each side. These basal

FIG. 33.

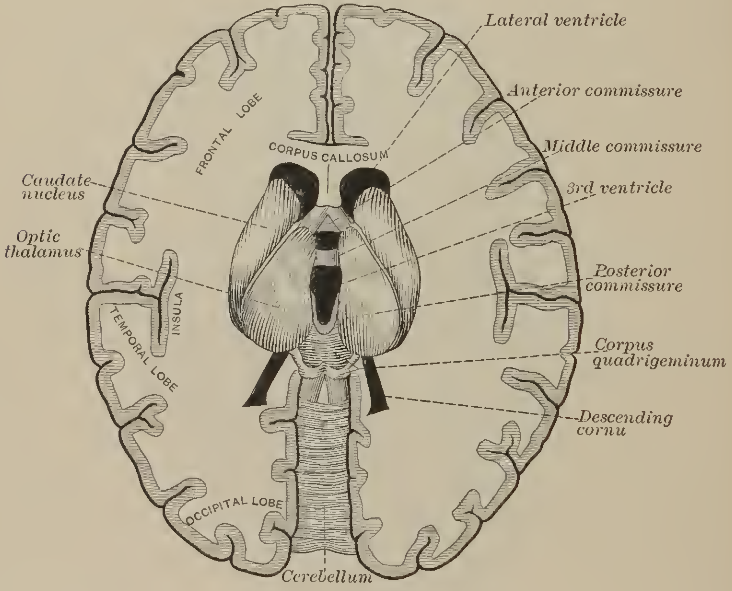


The centrum ovale and the corpus callosum.

ganglia consist of several masses of gray matter. On each side of the median line is seen a large pear-shaped body, the optic thalamus. To the outside of this is a tail-shaped body, commencing anteriorly in a large club-shaped head, and running posteriorly to pass downward and inward and terminating in a tail. This, because of its shape, is known as the nucleus caudatus, or the caudate nucleus. When a transverse section is made through these basal ganglia at a still deeper level (Fig. 35) this caudate nucleus will have had the body of it between the head and the tail cut away so that only the head will be seen anteriorly, whilst posteriorly the tail alone will be visible. It is therefore evident that the caudate nucleus is arched,

like a tadpole, from its head to its tail, and it is this arch that is cut away by the section of which we are now speaking. At this deeper level there comes into view another mass of gray matter, the so-called lenticular or bean-shaped nucleus, to the outside of the caudate nucleus (*i. e.*, its severed head and tail) and the optic thalamus. This lenticular nucleus has three compartments, an outer, a middle, and an inner one, seen in Fig. 35. The caudate nucleus and the lenticular nucleus were regarded by the older anatomists as essentially one structure, and were known by the collective name of the corpus striatum. Running between the head of the caudate nucleus anteriorly and internally, the optic thalamus and the tail of the caudate nucleus posteriorly and internally, and the lenticular nucleus externally, is a mass of white fibres known as the internal capsule. This

FIG. 34.

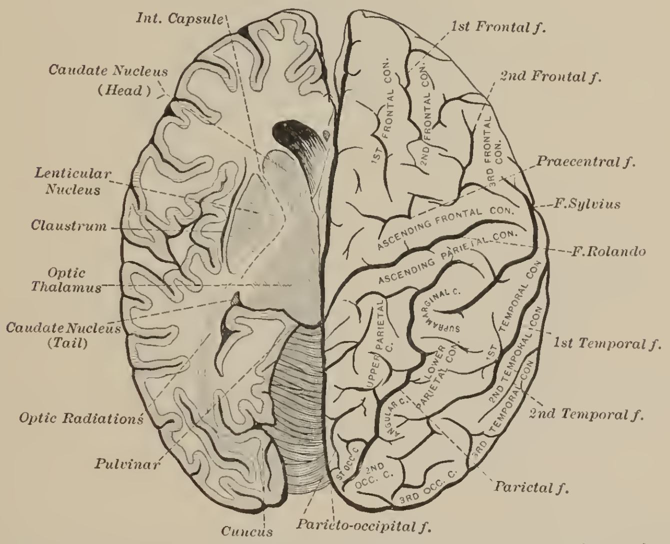


Basal ganglia of the cerebrum.

contains the nerve-fibres coming from the convolutions and converging at this point to pass in and between the deeper masses of gray matter. The nerve-fibres, therefore, which have been spread out over the wide surface of the overarching convolutions of the cortex, are here narrowed into the constricted strait of the internal capsule. This should be carefully borne in mind, because upon this anatomical fact depend some important points of diagnosis, as it will be evident that a small lesion capable of injuring many fibres which are packed together into the narrow compass of the internal capsule would be utterly incapable of injuring the same number of fibres if it attacked them after they had spread out over the wide area of the cortex. At the right side of the brain, in Fig. 35, for example, are figured the

different convolutions of the vertex of the brain. Let the reader contrast the size of the lesion that would be necessary to affect all the different centres upon these convolutions with the size of the lesion that could affect the nerve-fibres coming from these centres after they have converged into the internal capsule, and he will obtain a clear idea of what I am attempting to explain. This internal capsule is composed of an anterior portion and a posterior portion, and the point at which these bend into one another is known as the knee, or *genu*. The anterior portion contains the motor fibres, and the posterior portion the sensory fibres; indeed, it may be borne in mind as an invariable rule in tracing the course of a nerve-tract in the brain and cord, that the motor fibres lie always anteriorly, or toward the abdomen, or *ventrad*, as is technically said, whilst the sensory fibres always lie posteriorly, toward the back, or *dorsad*. Some of the fibres coming from the cortex pass straight through to lower levels, whilst others terminate in these basal ganglia, and others still originate in them and pass to the periphery or to other masses of gray

FIG. 35.



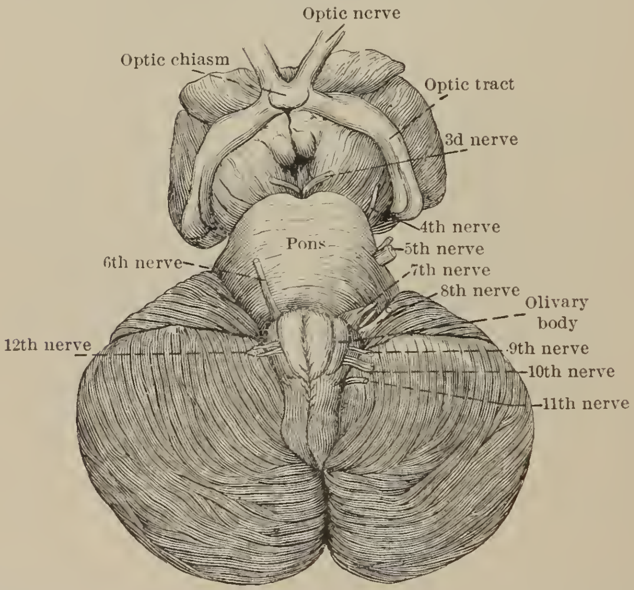
Convolutions of the vertex, on the right; on the left, the basal ganglia, the internal capsule, the centrum ovale, and the cuneus.

matter. A moment's thought will convince anyone that this is only what should be expected, because, unless the basal ganglia are to remain isolated, they must be connected with the cortex, with the periphery, and with other masses of gray matter. In that portion of the internal capsule lying anteriorly to the knee, or *genu*, are to be found the fibres coming from the frontal convolutions, whilst just about the "knee" are the fibres supplying the facial muscles, just behind these the fibres supplying the tongue, just behind these again the motor fibres going to the extremities, and still again just behind

these the sensory fibres. Entering the most posterior portion of the internal capsule is to be seen the so-called *optic radiation*, containing the fibres that come from the occipital lobe and enter the internal capsule, and then curve sharply inward in order to pass to the quadrigeminal and geniculate bodies, which we shall presently describe. Fig. 52 will show that the terminal branches of the Sylvian artery supplying the internal capsule supply these different regions, and that the arteries of the anterior portion are especially prone to hemorrhage, thus explaining the clinical peculiarity that hemorrhage into this region of the internal capsule is almost always productive of motor paralysis of the hemiplegic type, and that sensory impairment is usually slight or absent.

Outside of the lenticular nucleus (Fig. 35) is seen a strip of white matter, which is known as the *external capsule*, in the centre of

FIG. 36.

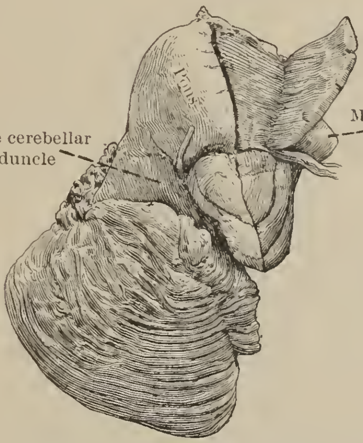


Pulvinar, optic nerve, optic chiasm, optic tract, pons, dorsal surface of medulla and upper portion of cord, base of cerebellum, and superficial origins of the second, third, fourth, fifth, sixth, seventh, eighth, ninth, tenth, eleventh, and twelfth cranial nerves.

which is a narrow streak of gray matter, the *claustrum*. External to the external capsule are those inturned convolutions lying buried in the fissure of Sylvius, and known as the *island of Reil*. The right side of Fig. 35 will make it clear how it is that the island of Reil comes in at this point. From the internal capsule the fibres pass on down into the *crura cerebri*, which can be seen at the base of the brain by going back to Fig. 18, where it will also be observed that their fanciful name of the *legs of the brain*, given them by the early anatomists, was not so badly chosen. The crura cerebri, however,

are more distinctly shown in Fig. 36, and this shows their relation to the optic chiasm and the surrounding under-lobes of the cerebrum. From the crura cerebri the nerve-fibres pass on through the *pons Varolii*, or the bridge of Varolius, which is the name given to the mass of fibres passing over from one lobe of the cerebellum to the other. Fig. 37 shows the transverse fibres cut in the centre so that one-half of the right side is lifted, in order to make clear the course of the long or longitudinal fibres. In this it will be evident how the nerve-fibres, after having traversed the pons, pass on down to the spinal cord, which is here called the oblong spinal cord, or *medulla oblongata*.

Fig. 37.



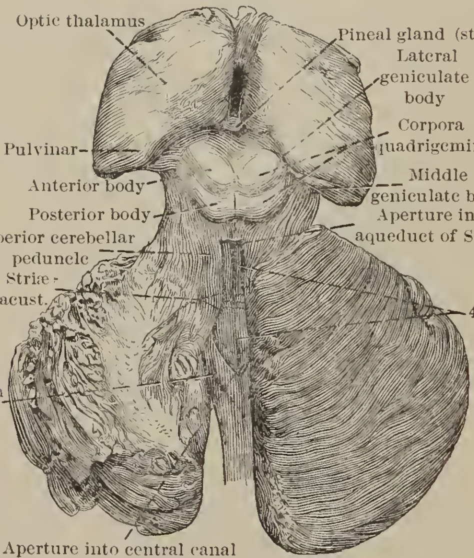
Specimen hardened in alcohol, showing the transverse and the longitudinal fibres of the pons.

This therefore brings us to the connection of most of the fibres with the spinal cord. Before passing on, however, to a study of this latter organ, we should have some understanding of certain regions lying above or dorsad of the crura and the pons.

Let us go back to Fig. 35. In this, as we have already seen, the optic thalamus and the two ganglia of the corpus striatum (the lenticular and caudate nucleus) are demonstrated. At the posterior and internal portion of the optic thalamus is an elevation of the body which is known as the *pulvinar*. Fig. 38 shows the relation of the optic thalami to certain other bodies, which it is important to study in relation to the optic nerve. Behind the pulvinar are seen four elevations of nervous tissue, which are known as the corpora quadrigemina, there being on each side an anterior and a posterior one. On the inner edge of the pulvinar, just about where the posterior quadrigeminal body joins it, is a little elevation known as the middle geniculate body, whilst a little further forward, just about where the anterior quadrigeminal body joins the pulvinar, is seen the lateral geniculate body.

The optic fibres, which we have seen in Fig. 34 coming into the posterior portion of the internal capsule, pass through the optic thalamus anterior to the pulvinar and into the anterior quadrigeminal body, and thence to the pulvinar, from which they go on down to the optic tract. The optic tract takes its origin in the anterior quadrigeminal body in a fine network coming from numerous delicate cells in what is known as the *stratum zonale*, as well as from the lateral geniculate body, the pulvinar, and a deeper-lying mass of gray matter with which we shall soon become acquainted, the *subthalamic body*, and also from the gray matter at the base of the brain, near

FIG. 38.



Specimen hardened in alcohol, showing pulvinar, pineal gland, corpora quadrigemina, geniculate bodies, superior cerebellar peduncle, fourth ventricle, aperture into aqueduct of Sylvius, aperture into central canal of spinal cord, restiform bodies, and striæ acousticæ.

the infundibulum. The function of the posterior quadrigeminal body is unknown, although Spitzka has shown that its extraordinary development in cetaceans and the large bands of fibres passing from it to the nucleus of the auditory nerve render it probable that it has some relation to hearing; and Baginski has demonstrated that it atrophies after destruction of the auditory nerve. We can perceive, therefore, that the course of the optic nerve is not so complicated as it would seem at first sight. From the anterior quadrigeminal body the optic nerve-fibres go to the cortex through the optic thalamus anterior to the pulvinar, into the posterior portion of the internal capsule, and thence to the occipital lobe as represented in Fig. 39. From this same point of origin in the anterior quadrigeminal body and the neighboring parts that have been men-

tioned, the fibres going to the retina first pass to the lateral geniculate body, then to the pulvinar, and then to the optic tracts, which, crossing in the optic chiasm, pass to the retina. Fig. 39 is a diagram of this course of the optic nerve. It shows its cortical origin in the occipital convolutions, thence its passage into the internal capsule, through the optic thalamus into the anterior quadrigeminal body, and thence to the geniculate body, to the optic tract, to the chiasm, to the retina. It will be observed that the optic fibres proceed from the left cuneus to the left half of each retina, which would be the outer half of the left eye and the inner half of the right eye. As the rays of light, however, cross in the optic lens when they come from without to reach the retina, this blindness of the outer half of

Fig. 39.

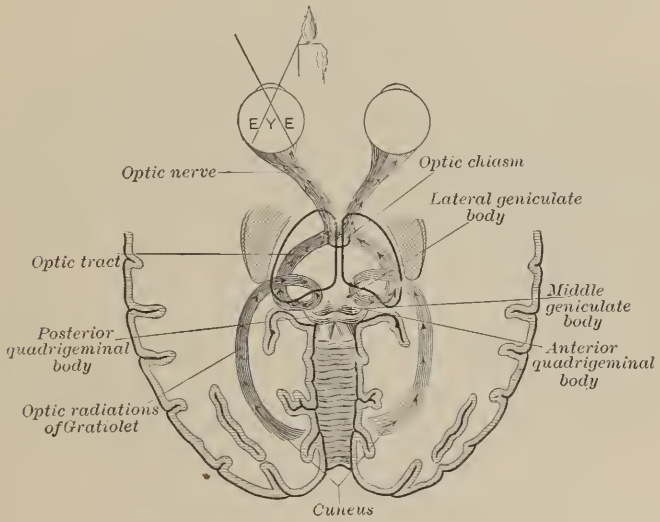


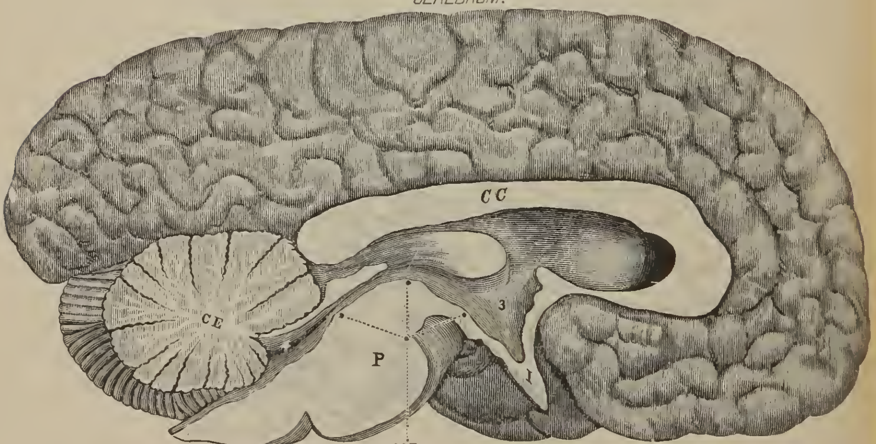
Diagram of the course of the optic fibres from the cuneus to the retina.

the left retina would incapacitate the patient from seeing objects to the inner side of the median line, whilst the blindness of the left or inner half of the right eye would incapacitate the patient from seeing objects to the outer side of the median line of the right eye. This should be clearly understood, for, as has already been stated, the blindness of the retina is called *hemiopia*, whilst the inability to see objects with this retina is called *hemianopsia*, so that the left hemiopia of the case which we have been supposing would cause a right hemianopsia.

Fig. 40 gives us another view of these central and basal ganglia which we have been describing. It represents a section midway through the corpus callosum (C C), which we have seen in Fig. 33 forming the floor of the great longitudinal fissure and connecting the two hemispheres. Beneath the corpus callosum is seen the third ventricle (3) running down into a funnel-shaped prolongation, known

as the infundibulum (I). Under the posterior half of the corpus callosum is seen the optic thalamus. Behind this are the corpora quadrigemina. Underneath the corpora quadrigemina is a narrow channel, the aqueduct of Sylvius, running back to connect with the fourth ventricle (4), which is prolonged into the central canal of the spinal cord. It will thus be apparent that the roof of the aqueduct of Sylvius is formed mainly by the corpora quadrigemina, and that from these bodies there runs to the cerebellum (C E) a thin strip of nervous tissue, known as the *velum medullare anterius*, or *valve of Vieussens*. It will therefore also be seen plainly that the roof of the fourth ventricle is formed partly by this valve of Vieussens, and partly by a lobe of the cerebellum, the lingula. The floor of the aqueduct of Sylvius and the floor of the fourth ventricle are formed by the posterior

FIG. 40.



Vertical section of specimen, showing corpus callosum, lateral ventricle, third ventricle, infundibulum, aqueduct of Sylvius, fourth ventricle, upper portion of central canal of spinal cord, corpora quadrigemina, velum medullare anterius, pons, medulla oblongata, cerebellum, and cerebrum.

surface of the pons (P) and medulla oblongata. Let us turn back for a moment to Fig. 36, so as to get the view of the under portion of this same pons, and to Fig. 37, so as to get an idea of the course of the transverse and longitudinal fibres of the pons, and then in our imagination let us transfer the pictures of Figs. 36 and 37 to the pons as it is represented in Fig. 40. Let us furthermore remember that the ventricle is a continuation of the central canal of the spinal cord, and that the aqueduct of Sylvius is the narrow aperture of connection between the third and fourth ventricles. All along the upper part of the central canal of the cord, on the floor of the fourth ventricle, along the under surface of the valve of Vieussens as it arches over the fourth ventricle and the aqueduct of Sylvius, along the floor of the aqueduct of Sylvius, and along the floor of the third ventricle, almost down to where it commences to narrow into the infundibulum,

is gray matter containing nerve-cells, and in this gray matter ten cranial nerves have their origin from the twelfth to the third inclusive, the twelfth most posteriorly and the third most anteriorly. In other words, it is evident that the spinal cord is thus prolonged into the brain, so that the medulla oblongata, as it is called, is nothing more than the upper enlarged end of the spinal cord, so altered in shape as

FIG. 41.

Section through the valve of Vieussens and the lingula. Magnified 6.6 diameters.

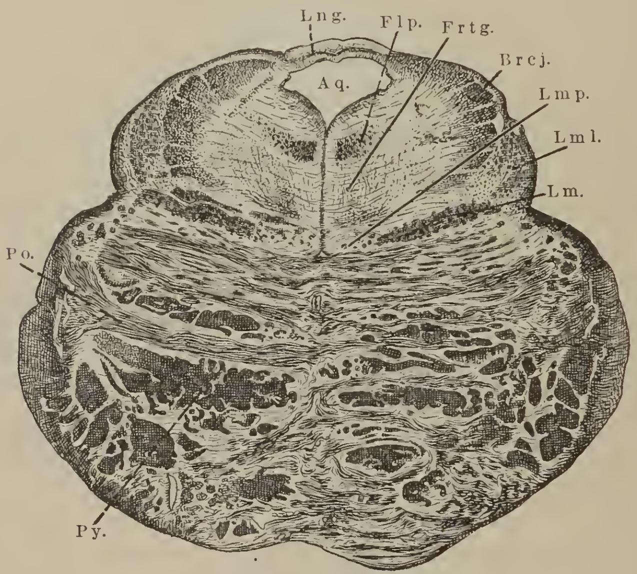
Py. Pyramid. *Po.* Transverse fibres of pons. *Lm.* Lemniscus. *Frtg.* Central field of tegmentum. *Flp.* Posterior longitudinal fasciculus. *Lco.* Locus cæruleus. *Niv.* Nucleus of the trochlear nerve. *Nvm.* Nucleus of motor root of fifth nerve. *Aq.* Aqueduct of Sylvius. *Lng.* Lingula. *Brcj.* Superior cerebellar peduncle. *Crst.* Restiform bodies. *Vm.* Fibres from motor root of fifth nerve.

to conform to the exigencies of cerebral architecture. Let us suppose that a number of microscopical sections are made through the regions of the pons, aqueduct of Sylvius, fourth ventricle, and upper part of the spinal cord, as shown in Fig. 40, and we will get an idea of the topography of this region by the study of them.

Fig. 41 represents a drawing of a section through the valve of

Vieussens and the lingula. In it is beautifully represented the course of the fibres going to the peripheral structures, the motor ones running in the pyramids at *Py*, and the sensory in the lemniscus at *Lm* and the tegmentum, *Frtg*, whilst the transverse fibres of the pons are seen at *Po*, so that we view here microscopically what we have seen with the naked eye in Fig. 37. The posterior longitudinal fasciculus, *Flp*, is a strand of nerve-fibres connecting the nuclei of the ocular muscles and probably also other motor nuclei; although there is still some doubt in the matter, for Spitzka's claim that this tract was well developed in amphibia and reptiles, unless their organs had been injured, has been contradicted by Von Gudden's assertion that moles,

Fig. 42.



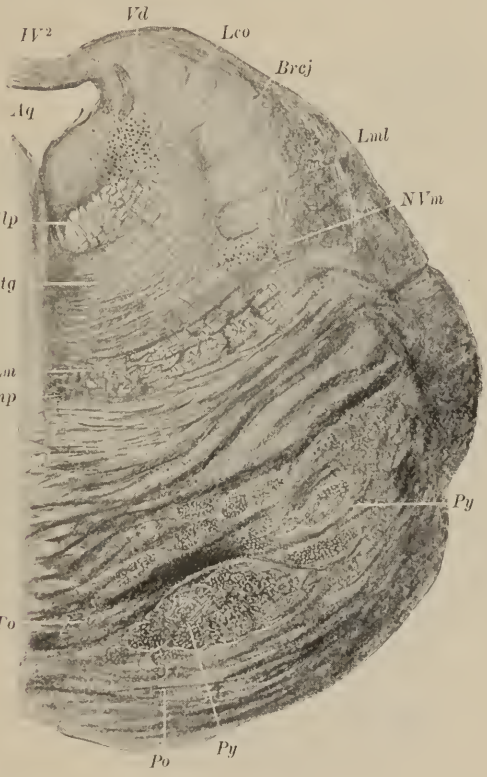
Section through the valve of Vieussens and the lingula, just behind the posterior quadrigeminal bodies. Magnified 5.2 diameters.

Py. Pyramid. Po. Transverse fibres of pons. Frtg. Central field of tegmentum. Lm. Lemniscus. Lml. Lateral lemniscus. Lmp. Nerve-tract connecting pes of crura cerebri with lemnisco-pedal tract. Flp. Posterior longitudinal fasciculus. Lng. Lingula of cerebellum. Aq. Aqueduct of Sylvius.

which have no nuclei of the nerves of the ocular muscles, possess as large a posterior fasciculus as rabbits. The nucleus of the motor root of the fifth pair is seen at *Nvm*, and at *Vm* are the nerve-fibres passing from it. To the outer side of these are the fibres of the sensory root of the fifth pair. At *Lco* is seen the so-called *locus cœruleus* or *substantia ferruginea*, a collection of large, dark, pigmented cells—although the former term, it should be remembered, is sometimes applied to a dark-brown or bluish spot, four to six millimetres in length, occasionally observed in the floor of the fourth ventricle. The *locus cœruleus* here depicted sends off fibres to the trigeminus,

these passing to the median line, to the raphe, then through the posterior longitudinal fasciculus, most of them reaching the motor branch of the trigeminus. At *Niv* is the nucleus of origin of the trochlear or fourth nerve. *Lng* is the lingula, the lobe of the cerebellum lying over the valve of Vieussens, which can also be seen in this illustra-

FIG. 43.



Section made just behind the posterior quadrigeminal bodies. Magnified 6.6 diameters.

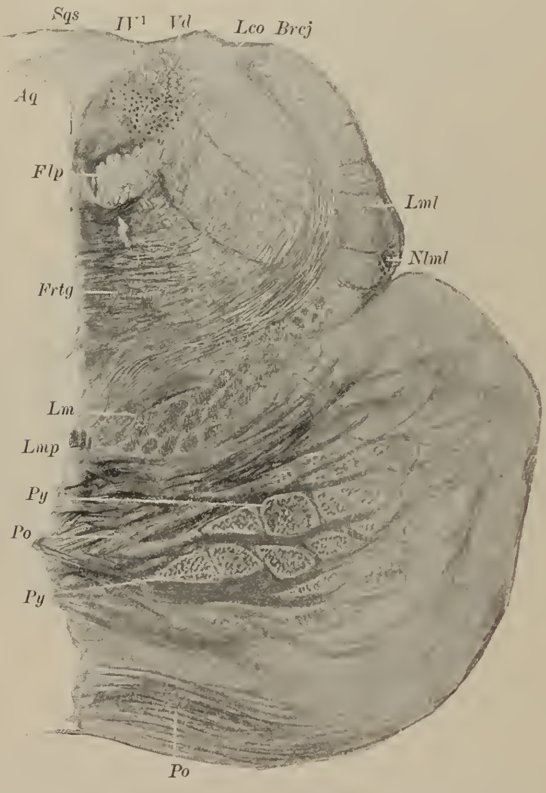
Py. Pyramid. *Po.* Transverse fibres of pons. *Lm.* Lemniscus. *Lml.* Lateral lemniscus. *Lmp.* Tract connecting lemuiscus and pes, lemnisco-pedal tract. *Frtg.* Central field of tegmentum. *Flp.* Posterior longitudinal fasciculus. *Lco.* Locus cæruleus. *Vd.* Descending root of fifth pair. *Aq.* Aqueduct of Sylvius. *IV²*. Decussation of trochlear nerve-fibres. *Brcj.* Superior cerebellar peduncle. *NVm.* Motor nucleus of fifth pair.

tion. *Brcj* is the superior cerebellar peduncle, and *Crst* is the restiform body, forming the inferior cerebellar peduncle.

Fig. 42 is a microscopical section made higher up, so that it comes just behind the posterior quadrigeminal bodies, as represented in Fig. 38. In it the motor fibres running to the periphery are seen in the pyramids, Py, and passing between these from side to side are

the transverse fibres of the pons, Po. Dorsad of these is the lemniscus, Lm, and the lateral lemniscus, Lml, whilst at Frtg is the central field of the tegmentum. The superior cerebellar peduncle is seen laterally to the tegmental field at Brcj. Just above the tegmental field is seen the section transversely of the posterior longitudinal fasciculus, Flp. The aqueduct of Sylvius is very clearly shown at Aq, and above it is the lower portion of the lingula.

FIG. 44.



Section through the posterior portion of the anterior quadrigeminal bodies. Magnified 6.6 diameters.

Py. Pyramid. *Po*. Transverse fibres of pons. *Lm*. Lemniscus. *Lml*. Lateral lemniscus. *Nlml*. Nucleus of lateral lemniscus. *Lmp*. Tract from lemniscus to pes, lemnisco-pedal tract. *Frtg*. Central field of tegmentum. *Flp*. Posterior longitudinal fasciculus. *Lco*. Locus cærulus. *IV*[1]. Descending root of trochlear nerve. *Vd*. Descending root of fifth pair. *Brcj*. Superior cerebellar peduncle. *Aq*. Aqueduct of Sylvius. *Sqs*. Sulcus beneath the quadrigeminal bodies.

In Fig. 43 is represented a section also made at the posterior portion of the quadrigeminal bodies, and in it are again shown the relations of the various parts which we have been considering: the pyramids at *Py*, the transverse fibres of the pons at *Po*, the sensory fibres of the lemniscus and the lateral lemniscus at *Lm* and *Lml*, the central field

of the tegmentum, *Frtg*, the posterior longitudinal fasciculus at *Flp*, and the superior cerebellar peduncle at *Brcj*. Certain other structures, however, here come into view. *Vd* is the descending root of the trigeminus; *Lmp* the tract connecting the sensory fibres of the lemniscus with the motor structures of the crura—what I term the lemnisco-pedal tract—and at IV^1 are seen the fibres of the trochlear nerve crossing in the roof of the fourth ventricle.

Fig. 44 represents a section made through the posterior portion

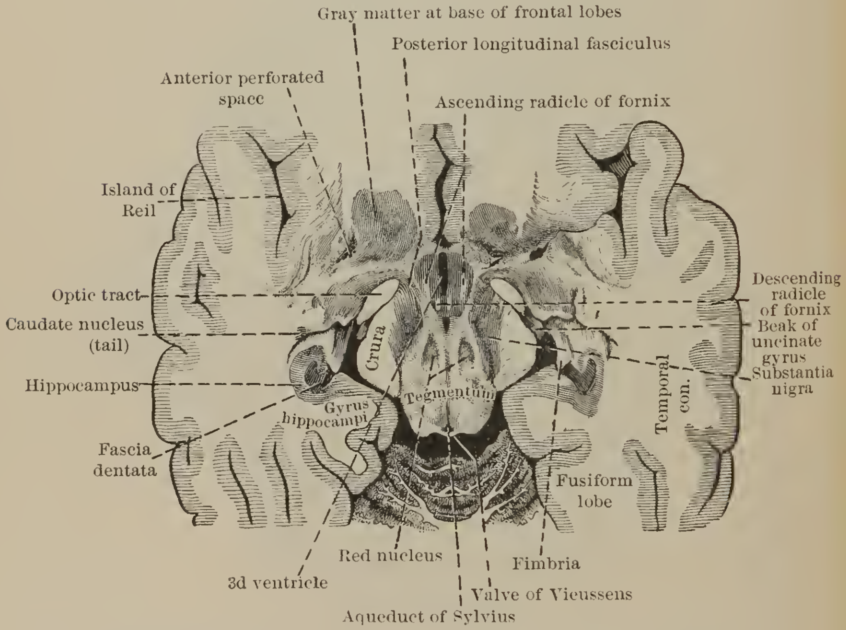
FIG. 45.

Pp. Pes or foot of the crura cerebri. *Sns.* Substantia nigra. *Csth.* Subthalamic body or body of Luys. *Ntg.* Red nucleus. *Al.* Ansa lenticularis. *F.* Forel's tegmental decussation. *M.* Meynert's ventral decussation. *N^3.* Nucleus of oculo-motor nerve. *Flp.* Posterior longitudinal fasciculus. *Aq.* Aqueduct of Sylvius. *Rsp.* Subpineal recess. *Cop.* Posterior commissure. *Vd.* Descending root of fifth. *QA.* Anterior quadrigeminal body. *Lm.* Lateral lemniscus.

of the anterior quadrigeminal bodies. The same structures can be studied in this as in Fig. 43, which we have just considered, except that the descending fibres of the trochlear nerve are distinctly represented at the side of the aqueduct at IV^1, whilst at *Sqs*, above the aqueduct of Sylvius, is seen the sulcus between the anterior quadrigeminal bodies, the so-called *sulcus corporum quadrigeminorum sagittalis*, and at *Nlml* is the nucleus of the lateral lemniscus.

Fig. 45 is a section through the region of the pineal gland, as represented in Fig. 38, exclusive of the pulvinar. Here we see that the motor and the sensory fibres are widely separated, the motor fibres lying in the so-called foot or pes of the crura cerebri at *Pp*, and the sensory above or dorsad in the lemniscus at *Lm*, the posterior longitudinal fasciculus at *Flp*, and at *Al* a set of fibres, probably sensory, that pass around the lenticular nucleus into the internal capsule, and which are therefore known as the *ansa lenticularis.* At N^3 is seen a portion of the nucleus of the hird or motor oculi nerve. At *M* is seen the tegmental decussation described by Meynert, and called after him, whilst at *F* is another tegmental decussation described by Forel, to which his name has

FIG. 46.



Transverse section of lowermost portion of cerebrum and basal ganglia, showing the main structures at this level. (DALTON'S *Topographical Anatomy of the Brain.*)

been given. At *Cop* is seen the posterior commissure; beneath it, at *Rsp*, the subpineal space, and above at the side, *QA*, the anterior quadrigeminal body. The descending root of the fifth pair is still seen at this level, as represented at *Vd*. But this figure beautifully represents three masses of gray matter which are coming to be of considerable importance in our modern anatomical knowledge. The *red nucleus* is represented at *Ntg* as a round body surrounded by fibres, whilst the *substantia nigra*, a collection of darkly pigmented cells, is seen at *Sns*, just above the pes of the crura cerebri, and at *Csth* is the *subthalamic body*, or *body of Luys*.

If we now go back, and from the superior aspect view this region

in which we have been making sections, we shall have such a picture as is presented in Fig. 38. Above we see the optic thalamus on each side, between them the stump of the pineal gland, just back of them the corpora quadrigemina, and still further down the fourth ventricle, a side view of which we have had in Fig. 40. At the upper end of the fourth ventricle is seen a narrow aperture passing into the aqueduct of Sylvius, and at the lower end of the fourth ventricle is another narrow passage into the central canal of the spinal cord. Turning back to Fig. 40 it will be readily perceived that the roof of the fourth ventricle is formed posteriorly by a portion of the cerebellum, and in front of this by the valve of Vieussens and the corpora quadrigemina, whilst the floor of the fourth ventricle consists simply, as has already been said, of the gray matter of the spinal cord which has come to the surface, instead of being buried in the centre, in the shape of horns, or cornua. In other words, the fourth ventricle is nothing more than a continuation of the central canal of the spinal cord.

In Fig. 46 is seen a horizontal section through the lowermost part of the upper portion of this region. It illustrates how the motor fibres come down in the crura cerebri, how the sensory fibres descend in the region of the tegmentum, and the relative position of the red nucleus and the substantia nigra. Contrast this with Fig. 45.

Fig. 47.

Horizontal section through the cerebellum. The section cut across anteriorly under the corpora quadrigemina. (From Stilling's *Atlas*.)

R, R. The superior cerebellar peduncles; between them the lingula. In front of this is seen the nucleus tegmenti (*m*) of the vermis, the nucleus globosus (*Ng*), the embolus (*Emb*), and the corpus dentatum of the cerebellum (*Cdc*), which are in the hemispheres on each side; at *Com* is the decussation of the anterior commissure, and at *Sem* is the semicircular fibres.

Now that we have seen the principal parts of the hemispheres, the basal ganglia, the pons and medulla, and have studied the details of these different figures, we are in a position intelligently to trace the course of the nerve-fibres through them to the periphery.

The cerebellum is an important organ which I have not as yet

studied, because I have thought it best to leave it until we had reached the present point and could understand the fibres coming into it from the structures of the pons and the subthalamic region. The cerebellum has been one of the mysteries of the nervous system, although Stilling, in his wonderful *Atlas*, embodied thirty years of research concerning it. Its outward shape is familiar to every physician, and we have seen it partially represented in Figs. 36 and 37. A horizontal section through it is shown in Fig. 47, which is taken from Stilling's *Atlas*. In the centre are seen the white substance of the vermis and the nucleus of the tegmentum, and anterior to these the decussation of the anterior commissure. On each side are the hemispheres, and in their white matter are seen the *nucleus globosus*, the *embolus*, and the nucleus dentatus. Between the superior peduncles, *R R* is the lingula, upon the valve of Vieussens. The

Fig. 48.

The three peduncles of the cerebellum. (From Hirschfeld and Sappey.)

1. Rhomboidal fossa of the fourth ventricle. 2. Striæ acousticæ. 3. Inferior peduncles, or restiform bodies. 4. Columns of Goll. 5. Superior cerebellar peduncles. 6. Lemniscus. 7. Middle peduncles. 8. Corpora quadrigemina.

cerebellum is connected with the cerebro-spinal nervous system by three peduncles:

1. The inferior peduncle, or restiform body, 3, Fig. 48.
2. The middle peduncle, or peduncle of the pons, 7, Fig. 48.
3. The superior peduncle, or the peduncle of the corpora quadrigemina, 5, Fig. 48.

The inferior peduncle or restiform body is composed of fibres from the direct cerebellar column of the cord which begin in the column of Clark and terminate in the vermis superior of the same side; of fibres arising from the nucleus of the column of Burdach, or the nucleus cuneatus (Plate II., *nucl. cun.*), and these also pass to the vermis superior; of fibres from the nucleus of the column of Goll of the same and opposite sides (Plate II., *nucl. grac.*), also going to the superior vermis; of fibres arising in the cells of Purkinje in the

cerebellar cortex and running to the lower olivary bodies of the opposite side; of fibres arising in the cells of the Deiters' nucleus and running to the nucleus tegmenti of the opposite cerebellar hemisphere, connecting the vestibular nerves and the cerebellum, and probably passing into the superior peduncles and thence to the cerebral cortex.

The middle peduncle is composed of fibres originating in the cerebellar cortex and running to the cells of the pons of the same and the opposite side, and thence onward to the cerebrum with the fibres of the antero-lateral column; whilst others cross in the raphe and go to those cells of the pons in which terminate the fibres coming down from the cerebral cortex of the frontal and occipito-temporal lobes, thus establishing a connection between the cerebellum and the cerebrum.

The superior peduncle obtains its fibres mostly from the nucleus dentatus and the cortex of the cerebellum, thence passing to the red nucleus of the opposite side; and from this red nucleus other fibres go to the optic thalamus, and from this to the cerebral cortex, probably to the central convolutions, thus establishing a second connection between the cerebrum and the cerebellum.

The cerebellum is an organ of co-ordination. It receives different sensations from the periphery and transmits them to the cerebrum, to whose cortex they bring images of space and position. From the cerebral cortex go other nerve-currents to the cerebellum, probably exercising a certain control or inhibition over co-ordination. These sensory impressions from the periphery are through the direct cerebellar column, which is thought to convey the visceral sensations; the fibres arising from the nuclei of the columns of Goll and Burdach possibly conveying the muscular sense; the so-called sensory cerebellar tract, running from the vestibular nerve to the nucleus tegmenti and bringing the cerebellum into connection with the circular canals of the labyrinth, which plays so important a part in the maintenance of equilibrium. The tracts conveying all these sensory impressions from the cerebellum to the cerebral cortex lie in the superior peduncle, whilst those through which the cerebral cortex exercises its inhibitions over the cerebellum are to be found in the middle peduncle. There is some doubt as to the course of the motor tracts direct from the cerebellum to the periphery, but it is probable that they are to be found in an indirect and a direct route, the former lying in the fibres running from the cerebellum to the motor zone of the cerebral cortex, although there is a variance of opinion as to whether these are in the superior or middle peduncle; and the latter in the fibres passing from the cerebellum through the inferior peduncle or restiform body to the spinal cord, where they may surround the cells in the anterior horns. Kölliker supposes that these impulses go through the fibres running from the cerebral cortex to the lower olivary bodies of the opposite side, and thence through the lateral columns of the cord to the anterior horns.

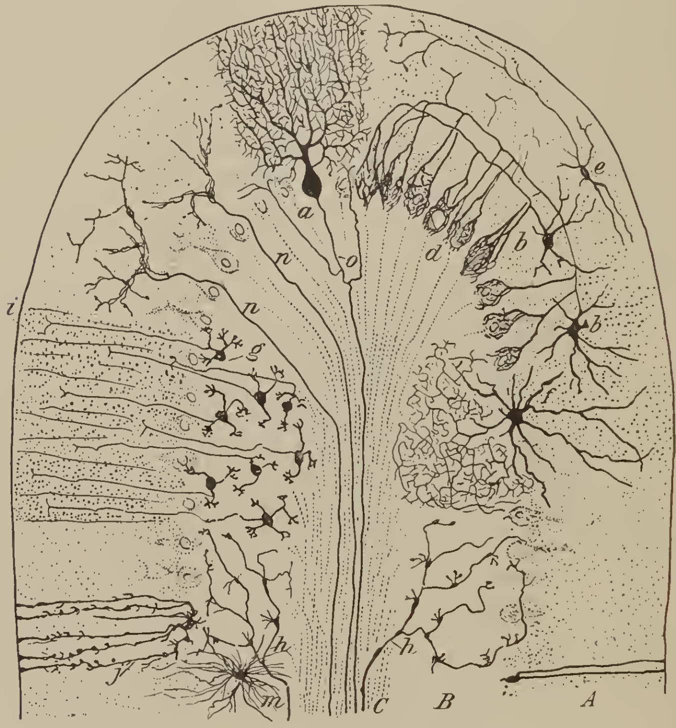
Our knowledge of the histology of the cerebellum has been much advanced by the studies of Cajal, Golgi, and those whom they have

incited. They have shown that the cortex of this organ is composed of three layers:

1. The superficial or molecular layer;
2. The granular layer, which is gray, yellowish, or reddish in color;
3. The zone of white substance.

The molecular layer contains two varieties of cells, those of Purkinje and the small star-shaped cells. The former are seen in Fig. 49, *a*. From the upper part of the cellular body proceed

FIG. 49.



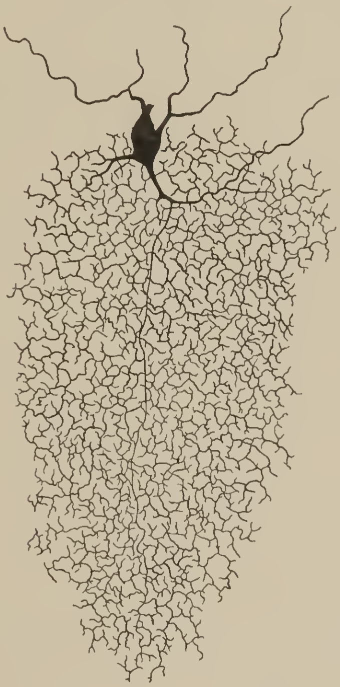
Semi-diagrammatic transverse section of a cerebellar convolution of a mammal. (CAJAL.)

A. Molecular layer. *B*. Granular layer. *C*. Zone of the white substance. *a*. Cell of Purkinje. *b*. Small star-shaped cell of the molecular layer. *d*. Terminal descending arborizations surrounding the cells of Purkinje. *e*. Superficial star-shaped cells. *f*. Large star-shaped cells of the granular layer. *g*. Granules with their ascending axis-cylinders bifurcated at *i*. *h*. Mossy fibres. *j*. Neuroglia cell with plume. *m*. Neuroglia cell of the granular layer. *n*. Climbing fibres. *o*. Ascending collaterals of the axis-cylinders of the Purkiuje cells.

one or several nervous trunks which, penetrating the molecular layer, expand into a flat and luxuriant arborization that is prolonged to the surface of the cerebellum. All the branches of this arborization terminate free, and have in their course a great number of collateral spines inserted perpendicularly. These arborizations are flat, and extend laterally, so that a longitudinal section along their axes shows them perfectlv. The axis-cylinder of these

cells of Purkinje takes on a covering of myelin immediately after leaving the cell, and descends to the white substance, emitting, however, at the level of two or three constrictions of the medullary layer, certain ascending collaterals which ramify in the inferior portion of the molecular layer and probably serve as means of connection between the different cells of Purkinje. A beautiful specimen of one of these cells is shown in Fig. 50. The star-shaped cells (*b*, Fig. 49) are small, and flattened transversely. Cajal claims to have discovered that they possess a very long axis-cylinder of an arched shape, which proceeds transversely, and that their descending collaterals, as well as the terminal arborizations of their axis-cylin-

FIG. 50.



Purkinje cell of the cerebellar cortex of a young cat. (VAN GEHUCTEN.)

ders, surround the cells of Purkinje, so that these latter bodies are, as it were, enclosed in a basket formed by the terminal ramifications, which are excessively thick and wavy. (*d*, Fig. 49.) To these structures Kölliker has given the name of the "end-baskets." The entirety of these fibres thus surrounding the cells of Purkinje is condensed at the inferior portion of the cellular body and envelops the axis-cylinder of the cell of Purkinje just at the point where the myelin is still lacking.

The granular layer is composed of very small bodies scantily supplied with protoplasm, and forming a very complicated mass beneath

the molecular layer. According to Cajal, their protoplasmic processes are short, two or three in number, and terminate in a finger-shaped arborization which appears to surround or touch the body of the neighboring granules. Their axis-cylinder is very fine, and ascends to the molecular layer, dividing in the form of a T at different levels of this zone, to become a longitudinal fibre which proceeds parallel to the cortex and therefore perpendicular to the arborization of the Purkinje cells. The parallel fibre does not give off any collateral in its course, but is prolonged to the limit of the lower layer of the cerebellum, where it terminates, almost at the white substance, by free and wavy thickening. Cajal has followed these parallel fibres throughout the small brains of the inferior vertebrates, reptiles, batrachians, and the fœtus of small mammals, and he believes that they connect the granules and the cells of Purkinje. It would thus appear that each granule may send a nervous current to all the cells of Purkinje situated in its radius and in the whole length of the cerebellar layer, or inversely, in the event that the nervous current should proceed in two directions, each cell of Purkinje can transmit its action to all the granules of the subjacent layer. The large star-shaped cells are found in the granular layer (Fig. 49, *f*). They are few in number, and their protoplasmic processes diverge in all directions, whilst their axis-cylinders split up into a great number of branches, which are lost among the granules. It is probable that these axis-cylinders terminate free.

The white substance of the cerebellum is composed of three varieties of nerve-fibres: 1, descending axis-cylinders coming from the cells of Purkinje; 2, thick nerve-fibres ascending and ramifying among the granules, the so-called *mossy* fibres; 3, thick fibres which ascend and ramify in the molecular layer, the so-called *climbing* fibres. The mossy fibres (*h*, Fig. 49) derive their name from the fact that they emit from place to place certain nodular thickenings resembling the moss of trees. At first they were considered as artificial productions, or *artefacts*, but their existence has been determined in all the vertebrata. They are found only in the granular layer, and terminate by free nodosities or by means of ascending moss-like expansions. Cajal thinks that they are possibly the continuations of the fibres from the direct cerebellar layer of the spinal cord. The climbing fibres (*n*, Fig. 49) are thick and medullated, either do not ramify at all, or but very little, and pass through the granular layer into the molecular, where they climb the ascending trunk of the cell of Purkinje and terminate in a wavy or plexiform arborization applied to the large primary and secondary branches of these Purkinje bodies. As yet, however, it is not known from what nerve-cells these climbing fibres arise.

According to Cajal, the protoplasmic arborizations and the bodies of all these cells transmit the nervous currents as well as their axis-cylinders, and this he believes because axis cylinders have been seen to ramify and terminate free upon these protoplasmic processes. He also maintains that a nerve-cell may have independent connections with other nerve-elements, inasmuch as in the cells of Purkinje each

portion of the body and of the protoplasmic arborizations is enveloped by an entirely different kind of nerve-fibre. Thus, the body is brought into relation, by means of the terminal baskets, with the star-shaped cells of the molecular layer; whilst the trunk of the main ascending branches have a connection, through the climbing fibres, with the elements of the spinal cord or the brain; and, still further, the terminal protoplasmic branches are brought into contact with the axis-cylinders of the granules, or the so-called parallel fibres. In most of these cases the connections are by *contiguity* and not by *continuity*, although it cannot as yet be absolutely stated that this is a universal law.

We are now in a position to try to unravel the course of the fibres from the spinal cord upward to the brain and the cerebellum.

The first peripheral station is formed by the cells of the ganglion upon the posterior root (*a*, *a*, Plate II. See also Fig. 5, B). The cells of these ganglia emit a nerve-process which splits into two, one going to the periphery (E, Fig. 11), the other passing to the spinal cord and forming, with other similar fibres, the posterior root (D, Fig. 11). These fibres of the posterior root pass into the spinal cord, and each one splits into an ascending and a descending one (Fig. 12). They proceed longitudinally in the posterior columns. All the descending ones are short (Plate II., 1′, 2′, 3′, blue), pass longitudinally a short distance downward, then bend at a right-angle and penetrate the gray matter, where they end in free nodules. The ascending fibres are divisible into short and long ones. The short ones (Plate II., 2, 3, blue) run for a short distance longitudinally upward, and then bend at a right-angle and pass into the gray substance of the cord, there to terminate in the usual free manner. The long ascending fibres (Plate II., 4, blue) pass longitudinally upward through the whole spinal cord, and, as with the others, bend at a right-angle and terminate with the usual free nodules in the gray substance of the medulla oblongata, in the region of the nucleus of the column of Goll and the nucleus of the column of Burdach, called respectively the *nucleus gracilis* and the *nucleus cuneatus*. All ascending and descending fibres give off collaterals in their course (Plate II., *col.*). These pass horizontally and penetrate the gray matter. It is thus evident that the terminals of all the descending and ascending fibres, together with their collaterals, pass into the gray matter of the cord; and as the terminations of the long fibres may be regarded as their final collaterals, it may be said that all the fibres coming into the cord penetrate the gray matter.

As these posterior root-fibres pass into the spinal cord they diverge and split into two portions—namely, a lateral and a medial. The lateral (Plate II., 2, blue) pass up longitudinally, and the most lateral of them constitute the Spitzka-Lissauer column, the ascending fibres being short. The medial portion (Plate II., 3, 4, blue) is much larger than the lateral, and consists of fibres which bend inward to constitute different portions of the column of Burdach, and here they first split up into ascending and descending fibres, the course of the

latter being unknown, whilst the former are divisible into short and long fibres. The short fibres (Plate II., 3, blue) bend into the gray matter after a longer or shorter course, whilst the long ones (Plate II., 4, blue) run toward the cerebrum through the whole course of the spinal cord and terminate in the medulla oblongata. These longer of the ascending fibres are pushed more and more in their course toward the middle line of the cord by the constant entrance of new fibres from the posterior roots (Plate II.); so that the long ascending fibres of the sacral nerves are pressed inward by the fibres entering the lumbar cord, these again by the dorsal, these by the cervical, and thus it comes to pass that in the cervical cord the sensory ascending fibres of the sacral nerves are most medial, whilst those of the cervical nerves are most lateral. The columns of Burdach contain mainly the short ascending fibres, although in them there are many long ascending fibres, which, however, are found nearest the median line, and they evidently pass over into the columns of Goll, through which they take their way to the medulla oblongata (Plate II., 4, blue). The columns of Goll consist almost exclusively of long ascending fibres, and they terminate in the nucleus of the column of Goll and the nucleus of the column of Burdach (Plate II., *nucl. grac.* and *nucl. cun.*), so that the cells of these nuclei are analogues of the column-cells of the cord. From these nuclei the sensory fibres proceed as follows: The axis-cylinder processes of their cells pass into the lemniscus (Plate II.), cross toward the median line, decussate with analogous fibres of the other side (so-called decussation of the lemniscus) and thus form the medial or upper lemniscus. Most of the sensory cranial nerves of the opposite side pass into this medial lemniscus and proceed with it to the cerebral cortex (Plate II.), the fibres of the medial lemniscus through the medulla oblongata, the pons, and the crus. In the latter they lie in the tegmentum. They then pass into the internal capsule in its posterior portion (Plate II., *caps. int.*), behind the motor fibres, and thence into the white matter constituting the corona radiata, and so on into the cortex, probably the ascending parietal convolution, the parietal lobe, and perchance also into the parietal, occipital, and temporal convolutions. All of these fibres give off collaterals in their course from the medulla oblongata to the cortex. Many of these do not reach the cortex, but end in different masses of gray matter on the way, such as the anterior quadrigeminal body, the optic thalamus, the lenticular nucleus, etc.

Besides this best known of the sensory paths, there are others through the anterior and lateral columns. Some of the nerve-fibres which have been described send collaterals to the column-cells of the spinal cord, and many of the short ascending and all of the descending fibres terminate in close contiguity to these same cells. The axis-cylinder processes of these column-cells become mainly the long fibres of the anterior and lateral columns. These in their turn give off collaterals to the gray matter of the cord, and finally bend at a right-angle to terminate, as do their collaterals, in the gray spinal substance. These longitudinal fibres compose the fundamental ante-

PLATE II.

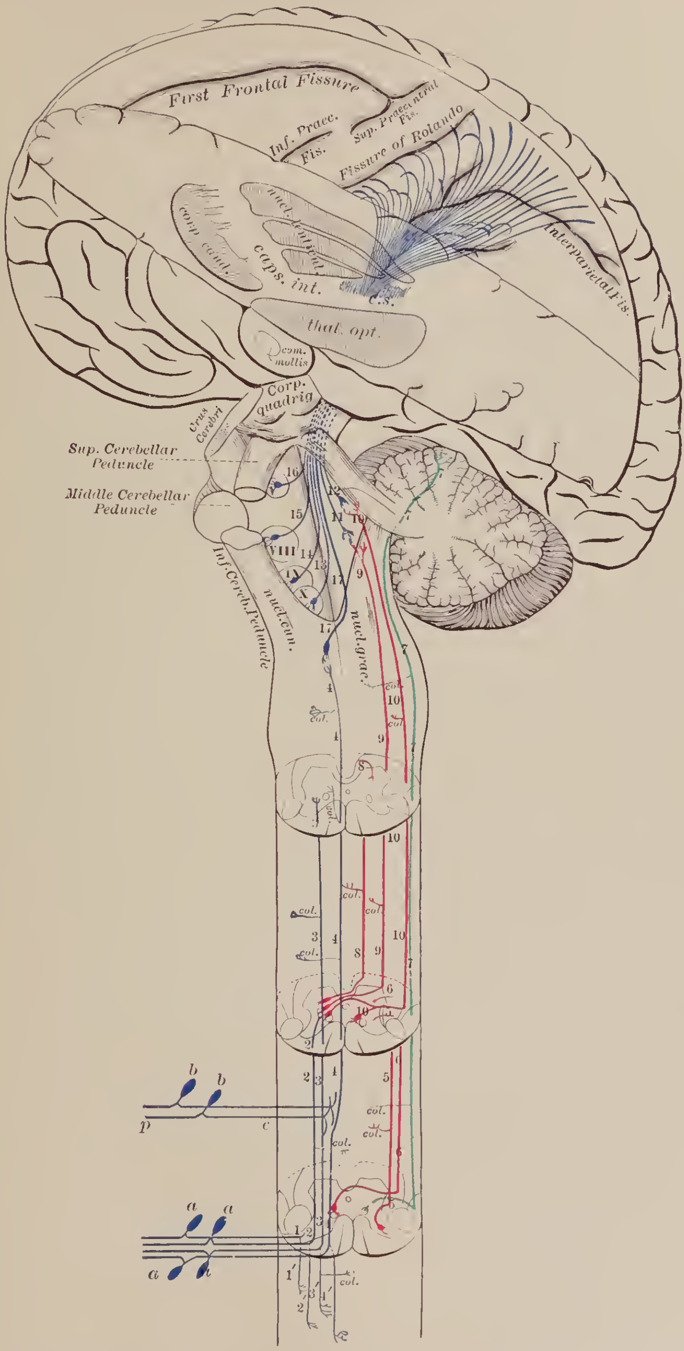


DIAGRAM SHOWING COURSE OF SENSORY FIBRES FROM PERIPHERY TO CORD, CEREBRUM AND CEREBELLUM.—(FLATAU.)

[To face page 78.

rior column, the fundamental lateral column, the Gowers, and the direct cerebellar column.

The anterior fundamental column is found throughout the spinal cord, and in the region of the pyramidal decussation it is pressed inward, but passes up from there as the so-called posterior longitudinal fasciculus. (See Figs. 41, 42, 43, 44, 45, *Flp*, and also Fig. 46.) The fibres of this fasciculus gradually end in the groups of cells of the medulla oblongata, pons, and subthalamic region, in the so-called gray reticular substance, finally terminating in the neighborhood of the nucleus of the third nerve and the anterior quadrigeminal body. In their course they give off collaterals to the motor nuclei of the eye-muscles and to the hypoglossal nerve, so that they are the principal means of communication for the reflexes from the sensory spinal nerves to the muscles of the eye and the tongue.

The lateral fundamental column is also found throughout the whole cord, and its fibres terminate in the cell-groups of the gray substance, especially in the nucleus reticularis tegmenti. It is probable that they end near the superior central nucleus immediately behind the posterior quadrigeminal bodies. In their course they give off collaterals to the motor nuclei of the fifth, seventh, ninth, tenth, and eleventh cranial nerves.

The column of Gowers first becomes visible in the lumbar cord, and the fibres are lost above in the neighborhood of the small olivary body.

It will thus be seen that the anterior and lateral fundamental column and the Gowers column have been followed into the cellular groups well up near the subthalamic region. These different cellular masses have been designated by Kölliker by the name of the *nucleus magnocellularis diffusus*, and it is possible that fibres proceed thence to the medial lemniscus and through this to the cerebral cortex. If this be true, the physiological importance of the lateral and anterior fundamental columns and the Gowers column would be thus explained: The nervous current coming from the periphery first passes, by means of the sensory fibres, to the column-cells in the gray matter of the cord. Thence go axis-cylinder processes which are either short, as in the anterior and lateral fundamental column; or long, as in the Gowers column, the direct cerebellar column, and individual fibres of the anterior and lateral fundamental column; or crossed, as in the anterior fundamental column and partially in the lateral fundamental column and the Gowers column; or uncrossed, as partially in the lateral fundamental column, the Gowers, and the direct cerebellar column. The longer of these fibres carry the sensory impulses from the column-cells to the cortex; first passing into the *nucleus magnocellularis diffusus*, which has been described above, and from there to the cortex through the medial lemniscus. These long columns decussate in the spinal cord in the anterior commissure. The short fibres proceed for only a short distance upward, give off collaterals, and end in the gray matter of the cord and the brain-stem. It is therefore possible that there is a direct and an indirect sensory path. The direct one consists of the peripheral fibre, the ganglion

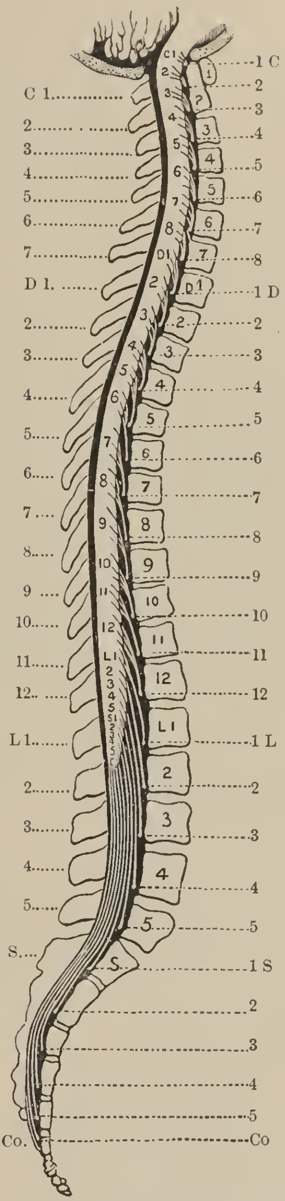
on the posterior root, the posterior root-fibre, the long ascending fibre in the posterior column, and the termination in the cells of the columns of Burdach and Goll (nucleus gracilis and nucleus cuneatus); thence, through the axis-cylinder processes of these cells, to the lemniscus, the medial lemniscus, the posterior part of the internal capsule, and the cortex. It has been supposed that this sensory path carries sensations of touch and muscular sense from the periphery to the cortex. The indirect sensory route lies in the anterior and lateral fundamental and the Gowers column, and consists of the peripheral nerve-fibres, the ganglion upon the posterior root, the fibres in the posterior column, the short ascending fibres, the collaterals of all the ascending and descending fibres, and the terminals near the column-cell; thence through the anterior commissure, the lateral and anterior fundamental and the Gowers column, to the cells of the nucleus magnocellularis diffusus; thence again through the medial lemniscus to the cortex. This indirect sensory path (Plate II., 11, 12, blue), possibly conducts the sensations of temperature and pain. It is also possible that the short longitudinal fibres form a part of this path, through the intercallation of the column-cells of the cord. It is thus evident that the direct and the indirect sensory paths undergo a decussation, the first in the brain-stem, the second in the spinal cord. The direct cerebellar column, however, does not decussate, and probably has no other function than to conduct impressions of coördination. The decussation of the indirect sensory path through the anterior commissure of the spinal cord has, however, been doubted by many authorities, and the question has not yet been positively decided.

We have seen that the ganglion upon the posterior root of the spinal cord contains cells which give origin to the sensory nerve-fibres proceeding inward. The fibres of the sensory cranial nerves take their origin in the same manner from cells outside the brain. From them the fibres of the sensory cranial nerves proceed to the cerebrum, where they—like the posterior root-fibres—divide into ascending and descending fibres, which have collaterals and terminals, the latter surrounding the sensory nuclei with their free ends. Thus the cells of these sensory nuclei are analogous to the column-cells of the gray matter of the spinal cord and those in the nuclei of the columns of Burdach and Goll. The axis-cylinders of these cells are either long or short The long ones decussate in the raphe and pass to the cortex. The short ones terminate in different cells of the gray cerebral substance and probably conduct the reflexes.

The course of the motor fibres from the cortex to the periphery is much simpler than the sensory highways which have just been described. The motor impulses proceed from the pyramidal cells of the motor area of the cortex to the pyramidal fibres, passing through the corona radiata to the motor nuclei of the cranial and spinal nerves, the former lying in the brain-stem, the latter in the anterior horns of the spinal cord. The axis-cylinder processes from the cells of these motor nuclei constitute the fibres of the peripheral motor nerves, and end free in the muscles. The beginning of the

motor path is in the large pyramidal cells of the motor area of the cortex, which is composed of the ascending parietal and ascending frontal convolutions, the bases of the three frontal convolutions and the paracentral lobule (Plate III., 1, 1, 1, etc.). Hence the nerve-fibres proceed through the white matter of the centrum ovale, or the so-called corona radiata, into the internal capsule (Plate III., 2, 2, 2, etc., *caps. int.*). From this point the motor fibres pass into the crura cerebri, lying most ventrad in what is known as the *pes*. Then they go into the pons and the medulla oblongata, where they are visible as two compact bundles, so-called pyramids (Plate III., *Py.*), and these pyramids can be followed to the lower end of the medulla oblongata.[1] At this point, in the region of the first and second cervical nerves, there is a decussation which extends over a distance of about six millimetres, and the pyramidal fibres split into two portions, one decussating and the other not. The decussating portion goes down the spinal cord in the lateral pyramidal column; the non-decussating part passes down on the same side in the anterior pyramidal column or column of Türck. The fibres of the lateral pyramidal column give off collaterals (Plate III., 3, 4, 5, *col.*, red), which proceed, at a right-angle from the main fibre, into the gray substance of the same side, where their terminal ends surround the cells of the anterior horn; and the main fibres themselves finally terminate in a similar manner. For these reasons the number of the lateral pyramidal fibres diminishes gradually downward until the column terminates in the lumbar enlargement at the region of the third and fourth sacral nerves. The fibres of the anterior pyramidal column also give off collaterals (Plate III., 6, 7, 8, *col.*, green) at right-angles, and these pass through the anterior commissure to the motor cells of the opposite

Fig. 51.



The mutual relations of the vertebral bodies and spines to the segments in the cord and to the exit of the nerves. (Gowers.)

[1] The student should follow the different columns of the medulla oblongata and the cord (Figs. 1, 2, 3, 41, 42, 43, 44, 45, and 46); and he had best do this in a second reading.

6

side, and the main fibres terminate in the same manner. This antero-pyramidal column ends about the lower third of the dorsal cord. It will be thus seen that these pyramidal fibres have two decussations, one in the region of the first and second cervical nerves, and the other at different levels of the cord through the anterior commissure. The axis-cylinder processes of the cells of the anterior horns pass into the anterior root-fibres of the same side and terminate free in muscles or glands. The motor cranial nerves all pass into this pyramidal tract at the knee of the internal capsule (Plate III., *x*, *t*, *tr*, *ab*, *f*, *g*, *h*, *v*), and they all decussate before reaching it. The course, however, of these cranial motor nerves from their respective nuclei to this point in the internal capsule has led to considerable differences of opinion among anatomists. It is an undoubted fact that many of the muscles supplied by these motor cranial nerves functionate bilaterally, as the muscles of mastication, of the eye, of mimic movements, and of the pupil, and for this reason it has been supposed that each motor cranial nerve receives fibres not only from its own nucleus, but also from that of the opposite side. But this belief has not yet met with complete anatomical confirmation.

In Fig. 51 is shown the relation of the various segments of the spinal cord to the bodies and spinous processes of the vertebræ. This is of importance from a surgical point of view, since the segments do not correspond to the vertebræ after which they are named. The following table is given by Starr, and has been modified by him recently, as the result of the analysis of a large number of cases, to show the localization of function in the different segments:

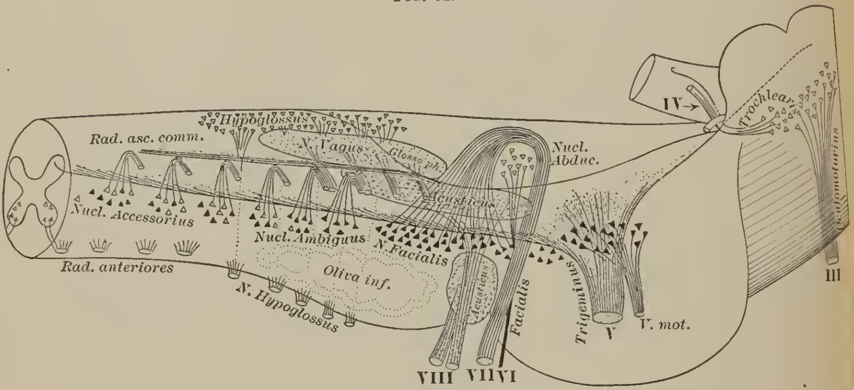
Segment.	Muscles.	Reflex.	Sensation.
II. and III. c.	Sterno-mastoid. Trapezius. Scaleni and neck. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex. Neck.
IV. c.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupil. 4th to 7th cervical. Dilatation of the pupil produced by irritation of neck.	Neck. Upper shoulder. Outer arm.
V. c.	Deltoid. Biceps. Coraco-brachialis Brachialis anticus. Supinator longus. Supinator brevis Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular. 5th cervical to first dorsal. Irritation of skin over the scapula produces contraction of the scapular muscles. Supinator longus. Tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm, front, and back.
VI. c.	Brachialis anticus. Serratus magnus. Triceps. Extensors of wrist and fingers. Pronators.	Triceps. 5th to 6th cervical. Tapping elbow-tendon produces extension of forearm. Posterior wrist. 6th to 8th cervical. Tapping tendons causes extension of hand.	Outer side of forearm, front, and back. Outer half of hand.

Segment.	Muscles.	Reflex.	Sensation.
VII. c.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Anterior wrist. 7th to 8th cervical. Tapping anterior tendons causes flexion of wrist. Palmar 7th cervical to 1st dorsal. Stroking palm causes closure of fingers.	Inner side and back of arm and fore-arm. Radial half of the hand.
VIII. c.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand, inner half.
I. d.	Extensors of thumb. Intrinsic hand-muscles. Thenar and hypothenar eminences.		Forearm, inner half. Ulnar distribution to hand.
II. to XII. d.	Muscles of back and abdomen. Erectores spinæ.	Epigastric, 4th to 7th dorsal. Tickling mammary region causes retraction of the epigastrium. Abdominal. 7th to 11th dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in horizontal bands running around and downward corresponding to spinal nerve. Upper gluteal region.
I. l.	Ilio-psoas. Sartorius. Muscles of abdomen.	Cremasteric. 1st to 3d lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. Over back above buttocks.
II. l.	Ilio-psoas. Sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patella tendon. Striking tendon causes extension of leg.	Front of thigh.
III. l.	Quadriceps femoris. Flexors of knee. Inner rotators of thigh. Abductors of thigh.		Front and outer side of thigh.
IV. l.	Abductors of thigh. Adductors of thigh. Tibialis anticus.	Gluteal. 4th to 5th lumbar. Stroking buttock causes dimpling in fold of buttock.	Outer side of leg. Inner side of thigh and leg to ankle. Inner side of foot.
V. l.	Outward rotators of thigh. Flexors of ankle. Extensors of toes.		Back of thigh, back of leg, and outer part of foot and leg.
I. to II. s.	Flexors of ankle. Long flexors of toes. Peronei. Intrinsic muscles of foot. Perineal muscles.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg. Foot-reflex. Achilles' tendon. Overextension of foot causes rapid flexion; ankle-clonus. Bladder and rectal centres.	Back of thigh, foot, outer side. Buttocks. Skin over sacrum, anus, perineum, genitals, and lower part of buttocks.

Before entering into the details of the origin of the different cranial nerves, Fig. 52 should be carefully studied. It is an excellent diagram from Edinger, showing the relative position of the nuclei, and it can be greatly elucidated by Figs. 36, 40, 41, 42, 43, 44, and 45.

The cranial nerves may be divided into the motor and sensory. To the former belong the oculo-motor, the trochlear, the abducens, the facial, the ninth, and the hypoglossal, as well as the motor portions of the three mixed nerves, the trigeminal, the glosso-pharyngeal, and the vagus. As with the motor spinal nerves, the tracts of each cranial one can be divided into a central and a peripheral portion. The central tracts begin in the large pyramidal cells of the motor area of

FIG. 52.



Relative location of the nuclei of the different cranial nerves. (EDINGER.)

the cortex, pass through the knee or *genu* of the internal capsule, thence downward to decussate and go to their motor nuclei, which are all in the brain-stem. (Plate III., IV., V., VI., VII., VIII., IX., X., XI., XII.) There is much divergence of opinion as to the exact peripheral tract of these cranial nerves. If one were to rely upon the physiological fact that the muscles supplied by the motor cranial nerves functionate bilaterally, as do the muscles of mastication, the eye, mimic movements, the pupil, etc., it might be supposed that each motor cranial nerve received fibres not only from its own nucleus, but also from the opposite one, or that the motor nuclei of the two sides were connected with each other. Anatomical investigations have not as yet, however, as has already been stated, confirmed either of these suppositions thoroughly.

The Oculo-motor Nerve, or the Third Pair, has its central tract from the cortex, from a point as yet not exactly determined in the human brain, thence to the internal capsule, and thence by decussating fibres to the cells of its motor nucleus. (Plate III., III.) Its peripheral tract is from the nucleus just beneath the anterior quadrigeminal bodies, downward, probably to split into two portions, one passing to the eye-muscles on the same side, and the other to those on the opposite, although the decussation has as yet only been demonstrated

PLATE III.

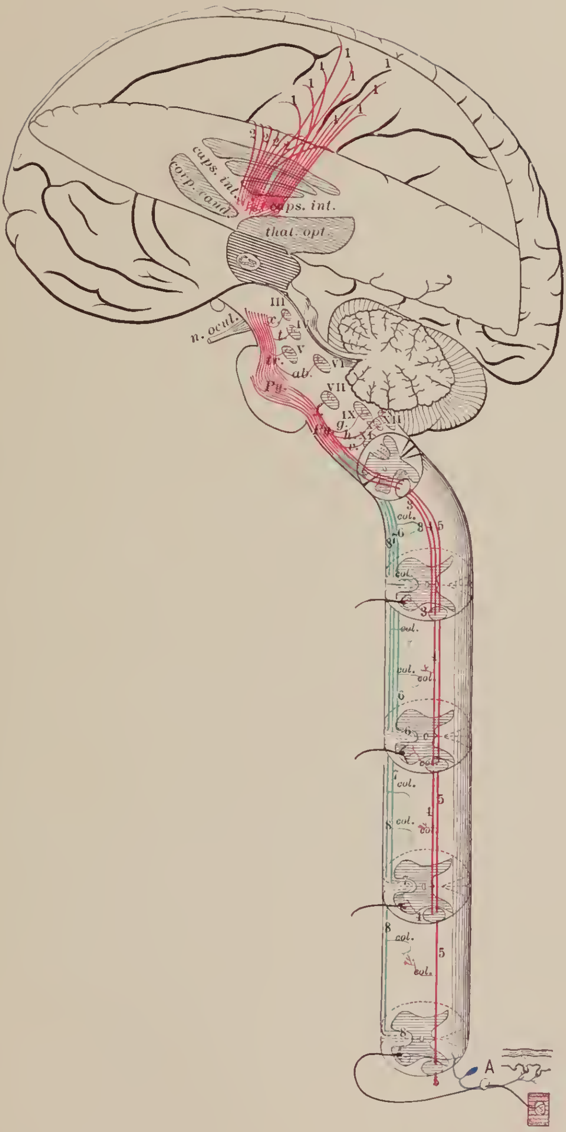


DIAGRAM SHOWING COURSE OF MOTOR FIBRES FROM THE CEREBRUM AND CORD TO THE PERIPHERY.—(FLATAU.)

[To face page 84.

for a portion of its fibres. Although the axis-cylinders of the cells of the nucleus pass over into the peripheral nerve-fibres, the collaterals are supposed to be surrounded by the central motor tract of the nerve, by the sensory fibres from the spinal cord through the posterior longitudinal fasciculus, and by the fibres of the optic nerve. The fibres from the spinal cord are presumably the medium of the reflex acts of the eye-muscles.

The Trochlear, or Fourth Nerve, has its central tract from an unknown point in the cerebral cortex to its nucleus. (Plate III., IV. *t*.) The peripheral tract is from the nucleus just beneath the posterior quadrigeminal bodies to a decussation shown in Plate III. It is uncertain as to whether fibres come from the other nucleus. Through its collaterals the cells of the nucleus of this nerve re-connect with its fibres going to the cortex, with fibres coming from the spinal cord, and with others from the optic.

The Abducens Nerve, the Sixth, has its central tract from an unknown point of the cortex to its nucleus. (Plate III., *a*, *b*.) This lies in the anterior portion of the floor of the fourth ventricle (Plate III., VI.), and its cells are connected with the cortical fibres, with sensory spinal fibres, and with fibres from the optic.

The Facial Nerve, the Seventh, has its central tract from the lower third of the ascending parietal and ascending frontal convolutions to its nucleus (Plate III., VII., *f*), which lies lateral and ventrad from the abducens nucleus at the lower end of the pons. (See Fig. 52.) It is well known that in cerebral hemorrhage producing what is known as hemiplegia only the lower branch of the facial is implicated, whilst that to the orbicularis oculi and frontal muscle are unaffected. The probable explanation of this lies in the fact that the motor nucleus for the upper branch is in the posterior portion of the oculo-motor nucleus of the same side, the axis-cylinders of whose cells pass into the fibres of the facial upon the same side, whilst the central tract connecting it with the cortex probably runs in the upper branch of the facial, which is not affected in most cases of cerebral hemorrhage. The cells of the motor nucleus are connected with the cortical fibres, with sensory spinal fibres, and with fibres from the trigeminal and cochlear nerves.

The Accessory Nerve of Willis, or the Eleventh, has its central tract from an unknown point in the cerebral cortex to its nucleus (Plate III., XI.) in the lowermost portion of the medulla oblongata. The posterior portion of the motor nucleus of the pneumogastric constitutes the motor nucleus of the cerebral portion of the eleventh nerve, whilst the anterior horn cells from the region of the fifth cervical nerve and somewhat upward constitute the motor nucleus of the spinal portion. The axis-cylinders of the motor nucleus pass into the fibres of the nerve on the same side. The roots of the eleventh nerve passing from the medulla oblongata are closely connected with those of the pneumogastric. The roots of the spinal portion of the eleventh pass from the side of the spinal cord, from the fifth cervical nerve upward. The cells of the motor nucleus are connected with the cortical fibres and with sensory spinal fibres.

The Hypoglossal Nerve has its central tract from the lower third of the ascending parietal and ascending frontal convolutions to its nucleus. (Plate III., XII., *h*.) This is about 18 mm. long, and stretches along the median sulcus on the floor of the fourth ventricle to the region of the *striæ acusticæ*. The nuclei on the two sides are connected by commissural fibres. It is probable that the fibres coming from one nucleus are joined by those from the other. The cells of this nucleus are connected with cortical fibres, with sensory spinal fibres, and with fibres from the sensory portion of the ninth and tenth nerves.

The Motor Portion of the Trigeminus, or Fifth Nerve, has its central tract probably from the lower third of the ascending parietal and ascending frontal convolutions to its nucleus (Plate III., V., *tr*.), on the upper portion of the floor of the fourth ventricle, as well as to certain motor cells which lie scattered in the lateral portion of the central gray matter up to the region of the anterior quadrigeminal bodies, and also in the so-called *locus cœruleus*. (Fig. 52.) The nerve-processes of the cells of the motor nucleus pass into the fibres of the motor trigeminal root, as well as into the descending root. These axis-cylinders are probably joined by others from the opposite nucleus. The nucleus is connected with cortical fibres, with sensory spinal fibres (the latter being the means of conduction of reflex movements, as in trismus, peripheral irritation, etc.), and with fibres of the sensory portion of the trigeminus.

The Motor Portion of the Glosso-pharyngeal Nerve, or the Ninth, has its central tract from an unknown point in the cerebral cortex to its nucleus (Plate III., IX., *g*), which is known as the nucleus ambiguus, lying fused with the motor nucleus of the pneumogastric on the floor of the fourth ventricle. (Fig. 52.) From the nucleus fibres pass to the peripheral nerve of the same side. The motor portion of the vagus or pneumogastric nerve, or the tenth, has the same tracts as the ninth. The cells of the motor nuclei of the ninth and tenth nerves are connected with cortical fibres, with sensory spinal fibres, and with fibres of the sensory portion of the trigeminus.

The tracts of the sensory cranial nerves are analogous to those of the spinal sensory nerves or the posterior roots. Just as these latter have their origin in the cells of the ganglia upon the posterior root, so do the sensory cranial nerves begin in cells lying outside the cranium, and from these the fibres pass into the cerebrum, where they divide, like the posterior root-fibres of the spinal cord, into descending and ascending fibres, which again split up into collaterals and end-fibres, the latter surrounding with their terminals the cells of the so-called sensory nuclei. The cells of these sensory nuclei are analogous to the column-cells of the spinal cord and to those of the nuclei of the column of Burdach and the column of Goll. The axis-cylinders of these cells are either long or short. The long ones decussate in the raphe of the medulla oblongata and carry their nerve-impulses uninterruptedly to the cortex, probably to the ascending parietal convolutions and the parietal lobe, thus constituting the central sensory tract. The short ones terminate in different cells of the

gray cerebral substance, and serve mainly to conduct the reflex movements.

The sensory portion of the trigeminal or fifth nerve is from the cells of the Gasserian ganglion through the sensory root to the sensory nucleus of the fifth pair (Plate II., V.) at the upper end of the floor of the fourth ventricle. Thence long fibres (Plate II., 16) decussate in the raphe to pass into the medial lemniscus and go with this to the cerebral cortex. The short ascending fibres go to the motor nuclei of the trigeminal, the facial, and the hypoglossal, and probably also to the motor portion of the ninth and tenth nerves, and serve mainly as media for the reflex movements. The sensory root-fibres and the fifth nerve split up into ascending and descending branches upon their entrance into the brain and stem. The descending branches form the tract known as the ascending trigeminal root (which is really the descending), whose terminals surround the cells of the sensory nucleus of the fifth and those of the posterior spinal cornua as low down as the region of the first cervical nerve.

The sensory portion of the glosso-pharyngeal and vagus nerve, or the ninth and tenth, arises as follows: The fibres of the ninth come from the jugular and petrosal ganglia, whilst those of the tenth have their origin in the jugular and cervical ganglia. This tract, by its terminals, surrounds the cells of the sensory nucleus (Plate II., IX., X.) lying to the side of the hypoglossal nucleus. From this long fibres (Plate II., 13, 14) ascend to decussate in the raphe and pass into the medial lemniscus, and thence to the cerebral cortex. Short nerve-fibres and collaterals go to the nucleus of the hypoglossal nerve, and probably are the media of the reflex movements. The descending fibres of the ninth and tenth nerves form the so-called solitary fasciculus, or the respiratory tract of Krause, and descend to surround by their terminals the cells of the sensory nucleus, as well as the cells in the posterior horns of the spinal cord as far down as the eighth cervical nerve.

The optic nerve is quite complicated in its tract. In the middle layers of the retina there are certain bipolar cells that are analogous to the cells in the posterior ganglia of the spinal cord; whilst in the deeper retinal strata are large ganglion-cells akin to the column-cells of the cord. From the bipolar cells axis-cylinders pass to surround the large ganglion-cells, and from the latter arise the fibres of the optic nerve. These decussate partially, as has been described, Fig. 39, in the chiasm, in such a way that fibres from the lateral or temporal half of the retina run in the lateral half of the chiasm, thence into the optic tract to the posterior portion of the internal capsule, and thence to the cortex of the cuneus, the occipital convolutions, and the lingual lobule of the same side. The fibres from the nasal or medial half of the retina—constituting the smaller portion of the tract—decussate in the chiasm, pass into the optic tract, thence into the posterior portion of the internal capsule to the cerebral cortex just described, but of the opposite hemisphere. These two central tracts are represented by the long ascending axis-cylinders of the large ganglion-cells of the retina. The shorter fibres end in the so-called primary optic

centres—*i. e.*, mainly in the lateral geniculate body, the anterior quadrigeminal body, the pulvinar, the medial geniculate body, and probably in the lenticular nucleus (Fig. 39), probably serving the purpose of the reflex movements. From these primary optic centres fibres pass to the cerebral cortex. The cells of the anterior quadrigeminal body, which are surrounded by the terminals of the short optic nerve-fibres, give off axis-cylinders that pass to the nuclei of the eye-nerves (the third, fourth, and sixth), which is a pathological and physiological fact of great importance.

The olfactory nerve takes its origin from certain bipolar cells of the nasal mucous membranes analogous to the cells of the spinal ganglia, as well as from the axis-cylinders of these bipolar-cells in the olfactory bulb, and which in their turn are akin to the column-cells of the cord. From these cells arise the fibres of the olfactory tract, a part decussating in the anterior cerebral commissure and passing to the gyrus hippocampi, the gyrus uncinatus, and the cornu ammonis of the opposite side; but the greater portion of the fibres do not decussate, going to the corresponding portions of the cerebral cortex. It is supposed that short fibres arise from these long ones to go to the cells in the olfactory tract, and these make possible the reflex movements.

The auditory, acoustic, or eighth nerve is extremely complicated. Near its entrance into the brain-stem this nerve divides into two roots surrounding the restiform body, namely, the lateral or outer root, the so-called cochlear nerve, and the medial or inner root, the so-called vestibular nerve. The former is most important to hearing, as the latter has nothing to do with audition, probably aiding in the perception of equilibrium. The fibres of the vestibular nerve arise in the ganglion of Scarpa, thence go through the medial root to surround the cells of the sensory nucleus (Plate II., VIII., 15), from which they proceed to decussate in the raphe, pass into the medial lemniscus and thence to the cortex. Another portion is very probably connected with the cerebellum. The cochlear nerve arises in the ganglion of Corti. Its fibres pass through the lateral root to surround the cells of the sensory nucleus, the so-called accessory and acoustic nuclei. The nerve-processes from the latter form the fibres of the acoustic striæ, whilst those of the former make up the so-called trapezoid body. The acoustic striæ pass dorsad, then bend downward toward the centre, to split into two portions. One goes to the upper olivary body of the same side, and thence to the lateral lemniscus. The other decussates in the raphe and goes to the olivary body of the opposite side, thence wending its way to the lateral lemniscus. There thus arises a partial crossing of the fibres of the acoustic striæ, as in a chiasm. The trapezoid body is composed of nerve-processes from the cells of the accessory nucleus. Its fibres pass to the medial line and divide into two portions. One immediately goes into the lateral lemniscus of the same side. The other passes to the raphe, and in its course its fibres are connected with the upper olivary body and the trapezoid nucleus of the same side, and ends in these nuclei, from which others pass to decussate in the raphe,

and thence pass to the lateral lemniscus. It will thus be seen that the lateral lemniscus is really the central auditory tract of the cochlear nerve, and is to be distinguished from the medial lemniscus, which constitutes a central pathway for the sensory spinal nerves of the fifth, ninth, tenth, and vestibular nerves. When this lateral lemniscus has reached the upper portion of the pons it divides into two portions, namely, the inner and the outer tract. The fibres of the inner tract end in the anterior and posterior quadrigeminal bodies of the same and the opposite side. They represent the short ascending fibres that serve mainly for reflex movements. The fibres terminating in the anterior quadrigeminal body are of especial importance, as they surround the large cells that are connected with the nuclei of the nerves of the eye-muscles, namely, the third, fourth, and sixth. These same cells are also surrounded by fibres from the optic nerve, so that they are centres for the optic and auditory nerves, and make possible the movements of the eye that result from optic and auditory sensations. The trapezoid body sends off collaterals in its course to the nucleus of the facial nerve, which serve the purpose of reflex movements, such as those of mimicry and the pointing of the ears in animals from auditory impressions. The collaterals to the central gray matter carry auditory impressions to the respiratory and vascular centres. The fibres of the outer bundle of the lateral lemniscus, joined by nerves from the posterior quadrigeminal bodies, pass into the posterior portion of the internal capsule, and thence to the cortex of the posterior portions of the two temporal convolutions. They belong to the long ascending fibres, and constitute the outer central auditory tract. It is possible, however, that shorter fibres may also connect different levels of nerve-ganglia with this portion of the cortex.

THE BLOODVESSELS OF THE CEREBRUM, CEREBELLUM, PONS, AND MEDULLA OBLONGATA.

Heubner and Duret have made most important researches into the vascular supply of the cerebrum, and have shown that there are two great sets of arterioles—one for the brain cortex, and the other for the brain stem, as the intra-cranial organs below the cortex and the centrum ovale are called, *i. e*, the basal ganglia and the sub-lying pons and medulla oblongata. The arteries of the brain stem arise at right-angles from the larger branches of the circle of Willis, and, entering the cerebral substance, branch in this without communication with each other. According to Heubner, they possess the peculiarity of taking their origin from the first portion of the large cerebral arteries as they pass over the white substance. These cerebral arterioles are beautifully shown in Fig. 53. From the short angle which is formed by the first portions of the Sylvian artery and the anterior cerebral artery, or the artery of the corpus callosum, arise only the vessels for the anterior half of the brain stem. From the anterior communicating branch of the anterior cerebral artery branches are given off to the head of the nucleus caudatus; from the point of the angle, the vessels

for the anterior limb of the internal capsule and the inner and middle segments of the lenticular nucleus; whilst from the lateral portion of the Sylvian artery are supplied the outer segment of the lenticular nucleus, the middle portion of the caudate nucleus, and the external capsule. The ganglia of the tegmental or dorsal portion of the crus are supplied from the commencing portions of the large arteries

FIG. 53.

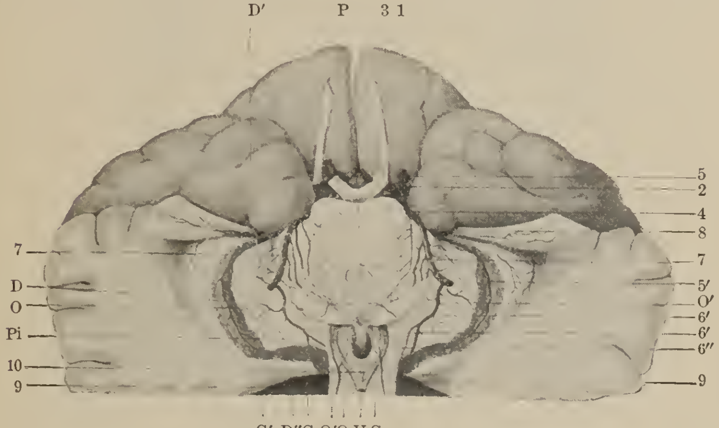
Branches of middle cerebral, anterior cerebral, and posterior cerebral. (DURET.)

1. Carotid artery. 2. Sylvian, or middle cerebral artery. 3. Anterior cerebral artery. 4. Posterior cerebral artery. 5. Arteries of the corpus striatum. 6. Anterior choroïdeal artery. 7. Posterior communicating artery. 8. Postero-lateral choroïdeal artery. 9. Postero-median choroïdeal artery. 10. Termination of callosal arteries. 11. Middle quadrigeminal arteries. 12. Postero-internal optic artery.

forming the circle of Willis. The choroïdeal artery and the posterior communicating supply the anterior, and the posterior cerebral the posterior ganglia. It must be borne in mind that although there is communications between these larger arteries at the base, there is none between the branches after they have plunged into the cerebral substance, and that these latter, therefore, are terminal arteries. The optic chiasm, the cranial nerves, the pons, the medulla oblongata, are thus supplied directly from the arteries of the circle of Willis without any intervention of the arteries coming from the pia mater. Duret has shown, furthermore, that many of these branches of the arteries of the anterior cerebral artery are inconstant. They

supply only the head of the caudate nucleus, and they produce very circumscribed lesions, which are the more dangerous because they may easily break into the ventricle. He has also shown that from the Sylvian artery there pass off the internal striated vessels going to the first and second segments of the lenticular nucleus and the internal capsule, and the external striated arteries, which are so called because they first supply the outer portion of the third segment and the lenticular nucleus, and then pass into the gray matter of the same and ascend. These latter branches are subdivided into

FIG. 54.



Arteries of the pons and the choroid plexus. (DURET.)

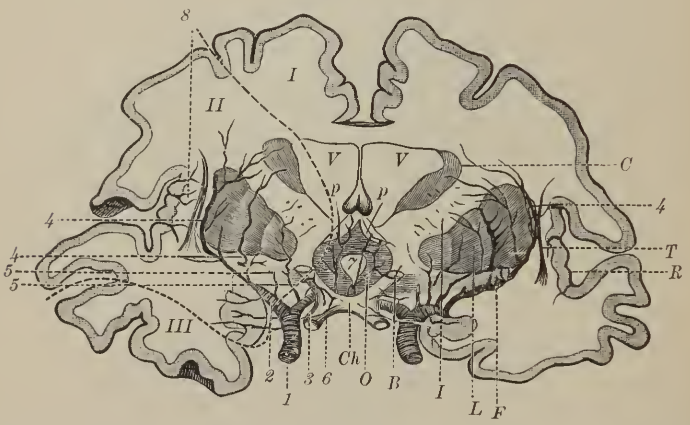
O. Part of optic thalamus not covered by choroid plexus. O′. Choroid plexus beneath the optic thalamus. C. Posterior portion of choroid, thrown back from its normal position above the crura cerebri and the quadrigeminal bodies. D. Choroid plexus. D′. Its anterior portion. D″. Where it curves to reach the lateral ventricles. G. Internal geniculate body. G′. External geniculate body. Q. Anterior quadrigeminal body. Q′. Posterior quadrigeminal body. P. Section of crura cerebri just above the pons. Pi. Pineal gland. 1. Carotid artery. 2. Sylvian, or middle cerebral artery. 3. Anterior cerebral artery. 4. Posterior cerebral artery. 5. Point of origin, from the carotid, of the anterior choroïdeal artery. 5′. Its course in the choroid plexus. 6. Postero-lateral choroïdeal artery. 6′. Its branch to the choroid plexus. 6″. Its branch to the velum interpositum. 7. Postero-external optic arteries, traversing the crura, near the geniculate bodies, to enter the optic thalamus. 8. Middle quadrigeminal artery, branching into the anterior and posterior quadrigeminal vessels. 9. Postero-median choroïdeal artery. 10. Termination of callosal artery in the pineal gland. V. Veins of Galen.

the lenticulo-striated vessels anteriorly, and the lenticulo-optic vessels posteriorly. The lenticulo-striated arteries are large, ascend into the third segment of the lenticular nucleus to the upper part of the internal capsule, and pass over to the caudate nucleus, terminating far forward in this. They are the most usual site of hemorrhages. The lenticulo-optic arteries pass through the posterior portion of the internal capsule, and into the anterior and outer portion of the optic thalamus. Duret has furthermore demonstrated that the posterior cerebral artery not only supplies the optic thalamus, but also the corpora quadrigemina and the tegmental region of the crus. One of these branches to the optic thalamus, the postero-internal optic artery,

supplying the inner surface, is prone to minute hemorrhages, which are very dangerous because of their tendency to break into the ventricle; whilst another branch, the posterior external optic artery, passes over the crus before reaching the hinder portion of the optic thalamus, and hemorrhages of it may therefore involve both the optic thalamus and the crus.

The arteries of the cortex are subdivided into secondary and tertiary branches within the pia, and from the plexus so constructed arise nutritive vessels which pass perpendicularly into the cerebral substance. (Fig. 56.) There is a difference of opinion between Heubner and Duret as to whether the areas to which these arterioles

FIG. 55.



Transverse section of the cerebral hemispheres, about 1 cm. behind the optic commissure.

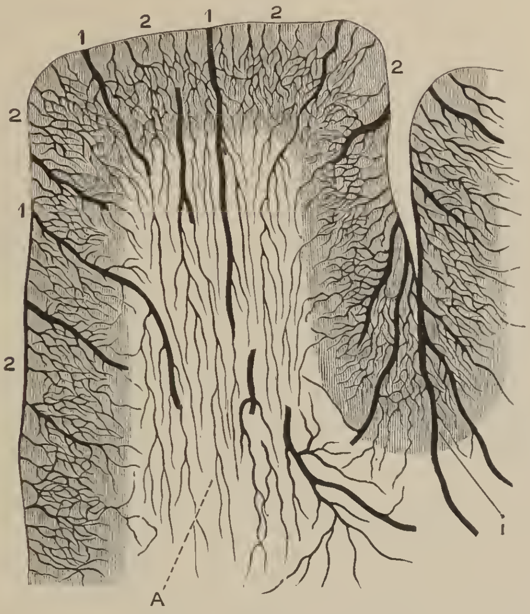
Ch. Chiasma. *B.* Section of the optic tract. *L.* Lenticular nucleus. *I.* Internal capsule. *C.* Caudate nucleus. *E.* External capsule. *T.* Claustrum. *R.* Island of Reil. *V, V.* Section of the lateral ventricle. *P, P.* Anterior pillars of the fornix. *O.* Gray substance of the third ventricle.

Vascular Areas: I. Anterior cerebral artery. II. Middle cerebral artery. III. Posterior cerebral artery.—1. Internal carotid artery. 2. Sylvian artery. 3. Anterior cerebral artery. 4, 4. External arteries of the corpus striatum (lenticulo-striate artery). 5, 5. Internal arteries of the corpus striatum (lenticular arteries). 6. Arteries of the chiasma. 7. Arteries of the commissure and anterior pillars of the fornix. 8. Arteries of the convolutions of the insula and claustrum. The opto-striate artery is not represented in the figure. (DURET.)

are distributed communicate with each other, Heubner contending that they are connected by small vessels of the calibre of a millimetre, whilst Duret maintains that each area is isolated. From the pia mater two sets of arteries pass into the cerebral substance: long or medullary vessels, and short or cortical ones. The long or medullary vessels, twelve or fifteen in number in the section of a convolution, pass vertically three to four centimetres deep, nearly to the end of the vessels coming up from the base, but without communicating with them; and it is in these badly nourished neutral territories between the two sets of arteries that certain senile softenings are especially to be found. The short or cortical vesels supply only the cortex and

the neighboring white matter. From them is formed the fine network in the cortex in the different layers of the nerve-cells. Through

FIG. 56.



Distribution of arteries to gray and white matter. 1, 1. Medullary branches passing directly through the gray matter to the white matter, and then terminating in branches that do not anastomose with their neighbors ("terminal arteries"). 1. Medullary arteries in the sulcus between two convolutions. 2, 2. Cortical arteries. (DURET.)

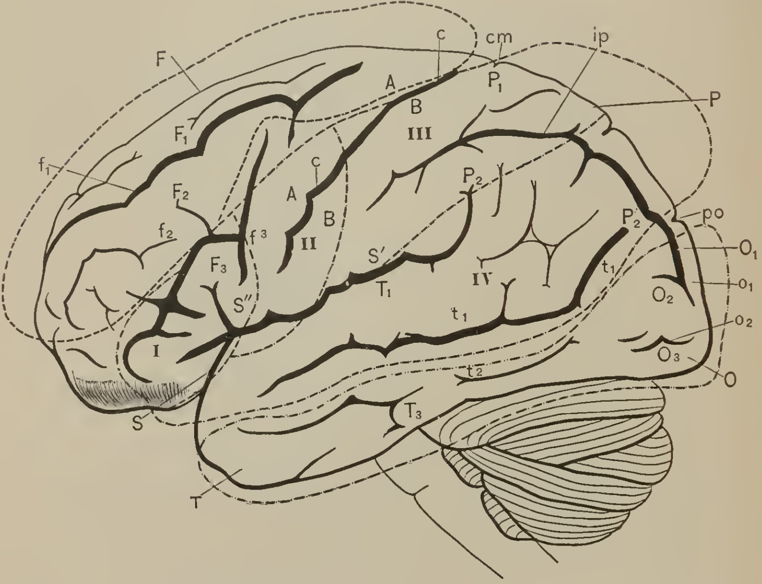
FIG. 57.

An artery of the cortex. Magnified 360 diameters.

obstruction of one of these arteries there will be formed a superficial softening of the cortex and the neighboring white matter, forming a

triangle whose basis will lie toward the surface. This is the so-called yellow softening (*plaque jaune*). In Fig. 57 is represented an artery of the cortex, with its peri-vascular space.

FIG. 58.



Outer surface of the left hemisphere. (After DURET.)

Distribution of Vessels.

The region bounded by the line (--------) represents the territory over which branches of the anterior cerebral artery are distributed.

The anterior regions bounded by the line (----------------) represent the territories over which branches of the middle cerebral artery are distributed.

I. Is the region of the external and inferior frontal artery.
II. Is the region of the anterior parietal artery.
III. Is the region of the posterior parietal artery.
IV. Is the region of the parieto-sphenoidal artery.

The posterior and inferior region bounded by the line (---- ----------------) represents the territory over which branches of the posterior cerebral artery are distributed.

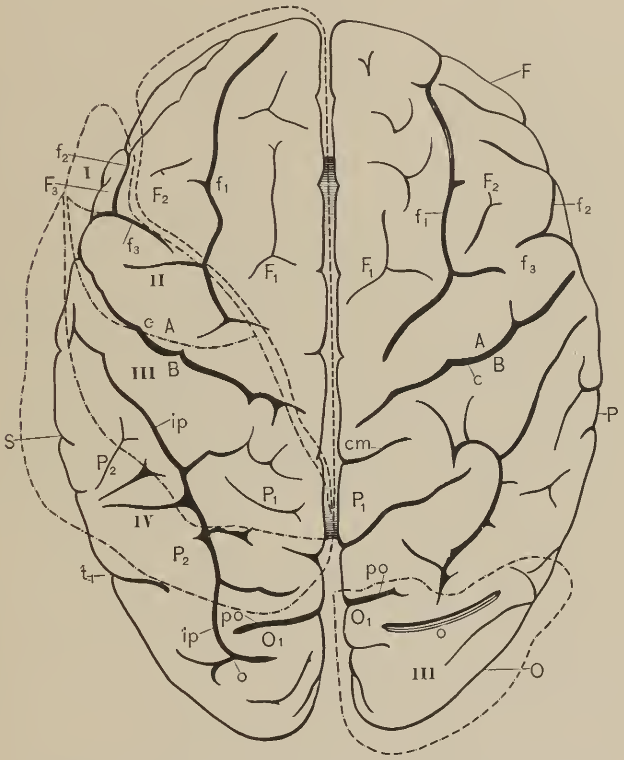
Fissures and Convolutions.

F. Frontal lobe. P. Parietal lobe. O. Occipital lobe. T. Temporo-sphenoidal lobe. S. Fissure of Sylvius; S′, horizontal; S″, ascending ramus of the same. c. Sulcus centralis or fissure of Rolando. A Anterior central or ascending frontal convolution. B. Posterior central or ascending parietal convolution. F^1, superior; F^2, middle; and F^3, inferior frontal convolutions. f^1, superior, and f^2, inferior frontal sulcus. f^3. Sulcus præcentralis. P^1. Superior parietal or postero-parietal lobule. P^2. Inferior parietal lobule, viz.: P^2. Gyrus supra-marginalis. $P^{2\prime}$. Gyrus angularis. ip Parietal fissure. cm. Termination of the calloso-marginal fissure. O^1, first; O^2, second; O^3, third occipital convolutions. po. Parieto-occipital fissure. o^1. Sulcus occipitalis transversus. o^2. Sulcus occipitalis longitudinalis inferior. T^1. first; t^2, second; T^3, third temporo-sphenoidal convolutions. t^1, first; t^2, second temporo-sphenoidal fissures.

According to Duret, the larger arteries of the cerebrum are only connected by the fine branches of longer or shorter vessels, and he

thinks that this is the reason that the areas of the central distribution of these arteries are much more prone to soften, whilst in the peripheral areas a distribution may, when the supply of blood from the true nutrient artery is cut off, be supplied by a collateral vessel.

FIG. 59.



Vascular areas of the upper surface of the cerebrum. (After DURET.)

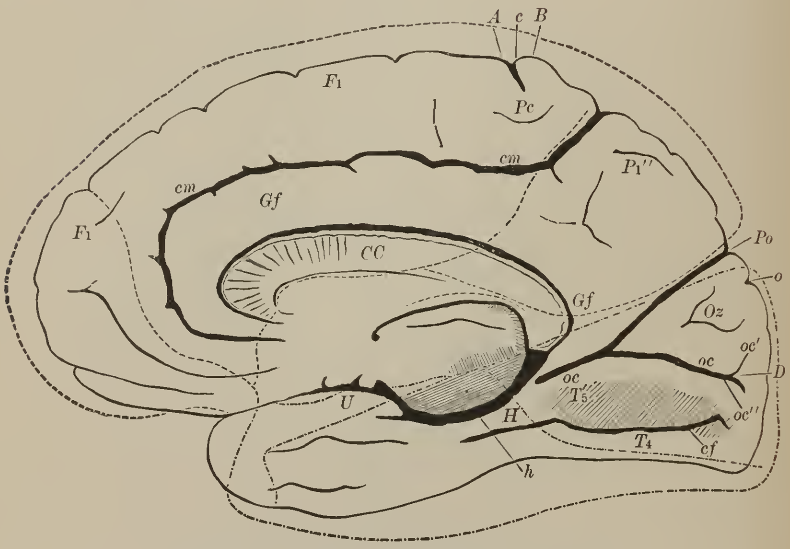
F. Frontal lobe. P. Parietal lobe. O. Occipital lobe. s. End of the horizontal ramus of the fissure of Sylvius. c. Fissure of Rolando. A. Ascending frontal convolution. B. Ascending parietal convolutions. F^1, F^2, F^3. Superior, middle, and inferior frontal convolutions. f^1, f^2. Superior and inferior frontal sulci. f^3. Sulcus præcentralis. P^1. Superior temporal lobule. P^2. Gyrus supra-marginalis. $P^{2'}$. Gyrus angularis. ip. Parietal fissure. cm. Calloso-marginal fissure. po, po. Parieto-occipital fissures. t^1. Superior temporal fissure. O^1. First occipital convolution. o. Transverse occipital sulcus.

Arteries: The line (.................) marks the limits of the distribution of the anterior cerebral; the line (- - - - - - - - - - - - -) on the left side of the figure limits the distribution of the Sylvian artery. I. External and inferior frontal artery. II. Anterior parietal artery. III. Posterior parietal artery. IV. Parieto-sphenoidal artery. The line (- - - -) on the right side of the figure limits the distribution of the posterior cerebral.

The Sylvian artery gives off four or five main branches, which pass over the island of Reil to reach the surface of the cerebrum. The first one supplies the first frontal convolution, especially in its rear portion, and hence arise the isolated softenings of Broca's

centre, which cause aphasia alone. The fourth branch supplies the lower parietal lobe and the upper portion of the first temporal convolution, whilst the fifth branch supplies the remaining portions of the first and second temporal convolutions. The anterior cerebral artery is divided into three branches, the first supplying the two convolutions on the under surface of the frontal lobe, the second going

Fig. 60.



Inner surface of the right hemisphere. (After Duret.)

Distribution of Vessels.

The regions bounded by the line (-----) represent the territories over which the branches of the anterior cerebral artery are distributed.

I. Is the territory of the interior and anterior frontal artery.

II. Is the territory of the internal and middle frontal artery.

III. Is the territory of the internal and posterior frontal artery.

The regions bounded by the line (--------) represent the territories over which the branches of the posterior cerebral artery are distributed.

II. Is the territory of the posterior temporal artery.

III. Is the territory of the occipital artery.

Fissures and Convolutions.

CC. Corpus callosum, longitudinally divided. *Gf.* Gyrus fornicatus. *H.* Gyrus hippocampi. *h.* Sulcus hippocampi. *U.* Uncinate gyrus. *cm.* Sulcus calloso-marginalis. F^1. Median aspect of the first frontal convolution. *c.* Fissure of Rolando. *A.* Ascending frontal; *B*, ascending parietal convolution. $P^{1\prime\prime}$. Precuneus. *Oz.* Cuneus. *Po.* Parieto-occipital fissure. *o.* Sulcus occipitalis transversus. *oc.* Calcarine fissure. *oc'.* Superior; *oc''.*, inferior ramus of the same. *D.* Gyrus descendens. T^4. Gyrus occipito-temporalis lateralis (lobulis fusiformis). T^3. Gyrus occipito-temporalis medialis (lobulus lingualis).

to the gyrus fornicatus, the corpus callosum, the second and third frontal convolutions, and the upper end of the ascending frontal convolution, whilst the third branch supplies the precuneus. The posterior cerebral artery splits into three branches. The first supplies only the hook-shaped end of the gyrus hippocampi, the second

supplies the fusiform lobule and the neighboring portion of the second temporal convolution, whilst the third branch nourishes the lingual lobe, the cuneus, and the occipital lobe.

Duret has shown that the pons and medulla are supplied by three different systems—the median, the radicular, and the indifferent vessels. The median arteries have a calibre of one-fourth to one-sixth of a millimetre, and arise from the basilar and the anterior spinal. They ascend perpendicularly in the raphe nearly to the gray matter, and supply the nuclei and the ependyma of the fourth ventricle, breaking up into fine networks for the different nuclei. They are terminal arteries. The anterior spinal artery is sometimes present on only one side, sometimes double, sometimes arising from each vertebral, but these branches generally unite in one stem. When there is only one, it generally arises from the left vertebral; if there are two, they are generally strongly anastomosed. The left vertebral is usually larger than the right. The radicular arteries arise from one of the larger branches that have been spoken of, and pass into the vessels that terminate on the nerve-roots in arterioles which have a diameter of one-third to one-fourth millimetre. These consist of ascending and descending branches, the latter passing to the nucleus, breaking up into a capillary network. It is therefore evident that each nucleus has two sources of arterial supply: one from the median and the other from the radicular arteries, the former being by far the most important. The indifferent arteries of the pons and medulla are supplied from the other larger vessels of the circle of Willis. The inferior cerebellar artery passes to the lateral portions of the medulla oblongata and the lower inferior cerebellar peduncle, and gives rise on each side to a posterior spinal artery which nourishes the posterior columns and finally divides into three branches: one for the vermis, anastomosing with its fellow of the opposite side and two lateral branches; one supplying the inner surface of the cerebellar hemisphere; the other the under surface of the same. These three arteries have enormous anastomoses on the surface of the cerebellum and also communicate with the posterior cerebral artery.

CRANIAL TOPOGRAPHY.

Of late years, since the localization of cortical centres has come so largely into play in neurological diagnosis, much attention has been paid to the relative position of the different fissures and convolutions of the brain and cerebellum and to the cranial sutures and bones. The subject was first studied in 1857 by Gratiolet, in 1861 and again in 1877 by Broca, and since then by Bischoff, Heftler, Turner, Féré, Fouillehouze, Ecker, Lucas-Championnière, Pozzi, Horsley, Thane, Hare, and Dana.

Broca has given certain names to the different points of union of the sutures, and these are marked out on the accompanying diagram, and with them the surgeon and neurologist should be familiar. The principal ones are the nasion, the inion, the glabella, the lambda, the stephanion, the pterion, and the asterion. The nasion is at the

junction of the nasal and the frontal bones; the inion is identical with the occipital protuberance; the glabella is a protuberance just above the nasion; and the asterion can be felt just behind the upper part of the mastoid process of the temporal bone; whilst the others are marked so plainly upon the diagram that they need no further description.

Fig. 61.

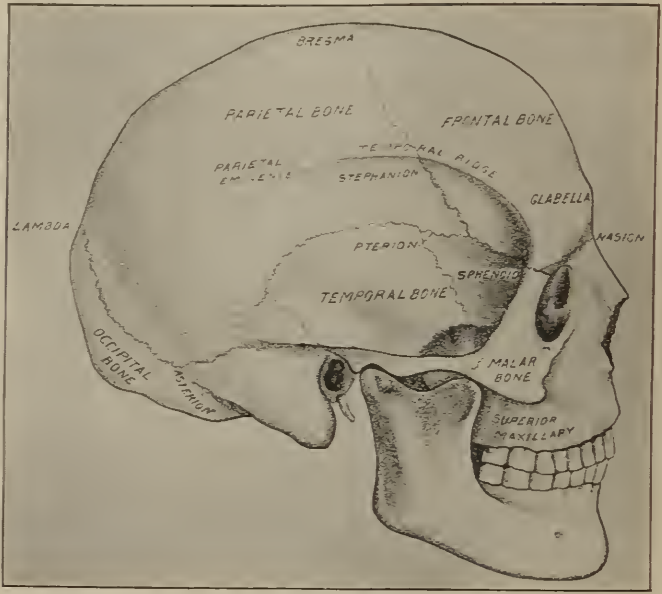


Diagram illustrating prominences of the skull.

The shape and various dimensions of the skull vary with age, sex, race, individuals, pathological conditions, and artificial deformities; but there is no great difference in the skulls of the European and the American races. The principal types of skull are the dolichocephalic, the brachycephalic, and the mesocephalic. The first is that in which the antero-posterior diameter is to the transverse as 100 is to 75 or less; the brachycephalic is that in which the antero-posterior diameter is to the transverse as 100 is to 80 or more; the mesocephalic is that in which the antero-posterior diameter is to the transverse as 100 is to 70 or to 80. The physiological limits in variation in the ratio of the antero-posterior to the transverse diameter are from 100 to 70, or 100 to 90. No great variations are found between the skulls of the two sexes, although in female skulls the bony ridges and protuberances are less marked, the inion less prominent, and the volume less, whilst the posterior half of the naso-occipital arc is greater. Nor has the shape of the skull been shown as yet to be of any particular importance among the different races, although it may be stated roughly that the English and Irish and

Scandinavians are dolichocephalic, whilst the negroes, Arabs, and Chinese are less long-headed in the ratio in which they are named; that the Germans, Russians, and Turks are brachycephalic; and

Fig. 62.

Photograph from Fraser, showing the cerebrum and the cerebellum in the cavity of the skull covered by the membranes; also the upper portion of the spinal cord.

that the American Indians, Dutchmen, and Parisians are mesocephalic. The Italian school have attempted to demonstrate the existence of a pathological or criminal skull, but they have not been successful.

Of much more practical value than all these variations of the skull are the relations of the different convolutions to the sutures. It must be borne in mind, however, that it is no longer necessary to define very minutely the relation of a suture to the skull-bone, be-

cause antiseptic surgery has rendered trephining nearly as harmless an operation as circumcision, and in most all cases the skull should be so liberally removed as to lay bare the whole cerebral lobe, or at least several convolutions. The approximate relation, therefore, of

FIG. 63.

Photograph from FRASER, showing the cerebrum, the cerebellum, and the upper portion of the spinal cord, the membranes having been removed.

the different cerebral fissures to the different sutures of the skull is all that it is necessary for the neurologist and the surgeon to know. Figs. 62 and 63, taken from Fraser's beautiful atlas, will give a true idea of the relative position of the main fissures and convolutions to

the skull, as the intra-cranial contents usually lie within the intra-cranial cavity. The fissures and convolutions can be readily identified by comparison with Fig. 14. Various instruments have been devised for marking off the skull, so-called cyrtometers, but an ordinary tape-line is as good as anything else. The rules following will be quite sufficient.

I. The longitudinal fissure corresponds with the median line of the vertex from the glabella, or junction of the nasal and frontal bones, to the inion, or occipital protuberance.

II. To find the fissure of Rolando: Measure the distance from the glabella to the inion, or occipital protuberance. Find 55.7 per cent. of this distance, and this will indicate the upper end of the fissure of Rolando, which is generally from 15.7 to 26.8 centimetres from the glabella. To find the course of this fissure, mark off from the upper end a line that shall form an angle of 67 degrees anteriorly with the longitudinal fissure. This will give the direction of the upper two-thirds of the fissure of Rolando, or about $2\frac{1}{2}$ inches, or 5.6 centimetres. The lower third of this fissure, about 2.1 centimetres in length, is somewhat more vertical, the total length of the whole fissure being 8.5 centimetres. To find the lower end of the fissure of Rolando, mark off a line from the stephanion to the asterion, and another from the bregma to the external auditory meatus, and the point of intersection of these two lines will be just over the lower end of the fissure, about one centimetre above the fissure of Sylvius. The upper end of this fissure of Rolando varies somewhat in its position in adults of different races and sexes, but the average variation is only about twenty millimetres, and the lower end has about the same variation. In idiots, whose cerebral growth has been impeded, the upper end is nearer the bregma, being only 30 to 32 centimetres behind it; but the lower end is not usually changed. In children the upper end of the fissure in the first months of life is from 30 to 35 centimetres behind the bregma, in the second and third years of life about 42 centimetres behind the bregma, and after this it rapidly approaches with every year to the distance of the adult brain.

III. The fissure of Sylvius: To find this draw a vertical line from the stephanion to the middle of the zygoma. Then draw a horizontal line from the external angular process to the highest point of the squamous suture. Prolong this latter line, gradually curving upward until it reaches the parietal eminence. The junction of the two lines will indicate the beginning of the fissure of Sylvius. The vertical line indicates approximately the position of the anterior or vertical branch of the fissure, whilst the posterior part of the horizontal line indicates the position of the posterior branch of the fissure. The third frontal fissure lies just anteriorly to the vertical branch of the fissure, *i. e.*, just anteriorly to the junction of the two lines that we have indicated. The tip of the temporal lobe is just back of the posterior edge of the orbital process of the malar bone. In children up to the third or fourth year the fissure of Sylvius is more oblique,

and lies somewhat more above the squamous suture than the lines which I have indicated.

IV. The parieto-occipital fissure: Measure the distance from the nasion, or junction of the nasal and frontal bones, to the inion or occipital protuberance, and take 22.8 per cent. of this, and this will indicate the position of the lambda. Its average distance from the occipital protuberance in adults is 7.42 centimetres. It must be remembered, however, that this parieto-occipital fissure is subject to great variation. Thus Heftler found it just above the lambda, Ecker several millimetres anterior, whilst Fouillehouze located it from 5 to 21 millimetres anterior; Féré found it under the lambda in 39 out of 62 cases, and a few millimetres above it in 24 cases; and Turner, Broca, Reid, Horsley, and Hare are not agreed. In children, too, this parieto-occipital fissure is from 5 to 31 millimetres in front of the lambda. In all cases, therefore, the occipital lobe should be generously uncovered.

V. The parietal fissure: The finding of this fissure is a somewhat complicated matter, if its exact site is to be determined from the skull. The fissures of Rolando and Sylvius and the parieto-occipital fissure should be marked out in the manner that has been described. The anterior end of the parietal fissure can then be found about one inch above and behind the angle formed by the fissures of Rolando and Sylvius, whilst the posterior part lies about one-half inch outside the parieto-occipital fissure. As this parietal fissure, however, is extremely variable, the best way to localize it is to uncover the parietal lobe, through the centre of which it runs.

VI. The precentral fissure and the first and second frontal fissures: These fissures can best be found by first ascertaining the position of the fissures of Sylvius and Rolando. The precentral fissure at its lower end is about 1 centimetre above the Sylvian fissure, whilst its upper end is about 2.5 millimetres anterior to the fissure of Rolando. The first frontal fissure begins at about 2.5 centimetres in front of the fissure of Rolando, running forward parallel to the longitudinal fissures. The second frontal fissure passes forward from the precentral fissure at a point a little above the stephanion, and continues its course under the frontal part of the temporal ridge.

VII. The temporal lobe and the first and second temporal fissures: The upper boundary of the temporal is the fissure of Sylvius and the lower boundary is a line drawn from a point slightly above the zygoma and the external meatus to the asterion, and continued along the occipital curve to the inion. The anterior border of the temporal lobe is about beneath the posterior edge of the orbital process of the malar bone. Bergmann states that a trephine placed one-half inch above the external auditory meatus would enter the lower part of the lobe, whilst the middle part is in the vertical line from the posterior border of the mastoid process. To find the first temporal fissure a line should be drawn about 2.5 centimetres below and parallel to the Sylvian fissure, whilst the second temporal fissure is about 2 centimetres below the first These are Reid's rules, and I believe they are reliable.

VIII. The occipital lobe and anterior occipital fissure can be found by curving a line from the lambda to a point about two-thirds of the distance between the inion and the asterion, and regarding this as the anterior border of the occipital lobe, whilst the superior occipital curved line may represent the lower border.

IX. The central ganglia of the corpus striatum and optic thalamus: Draw a line from the upper end of the fissure of Rolando to the asterion. This may be taken to be the posterior boundary of the optic thalamus. Draw a second line parallel to the first, slightly in front of the beginning of the fissure of Sylvius. This may be taken to be the boundary anteriorly of the corpus striatum. The upper surface of the ganglia will be about 45 millimetres below the surface of the scalp at the bregma.

HEAT-CENTRES.

Very little indeed is known about the heat-centres. Twenty-five years ago Tscheschichin found that a heat-centre existed somewhere above the pons Varolii in the brain. H. C. Wood had arrived at the same conclusion. Richet, in 1884, found that there was a heat-centre somewhere in the anterior part of the brain. In April, 1884, Isaac Ott located this heat-centre in the anterior and inner portion of the corpora striata of rabbits. In December of the same year Aronsohn and Sachs found that a puncture of the brain caused an increase of the temperature of the body. In July, 1885, Ott published a further communication upon the subject, stating that he had localized a heat-centre at the anterior and inner portion of the optic thalami. In October of the same year Sachs and Aronsohn again returned to the matter, localizing this heat-centre that they had previously described in the *nodus cursorious* of the optic thalami of the rabbit. Schreiber, Budge, Lussana, and Christiani had shown that various sections at the base of the brain and at the vertex were capable of causing rises of temperature; but it would seem as if the precise observations of Aronsohn, Sachs, and Ott had localized the heat-centre of which the other observers had merely seen the effects. Schreiber, for instance, found a marked increase in temperature after section of the medulla oblongata at its junction with the pons, whilst Budge had located vasomotor fibres in the crura cerebri. Schiff, Brown-Séquard, Ebstein, and others also showed that hemorrhages had been found in the pleura, lungs, stomach, intestines, and kidneys, after injuries of the corpora striata, the pons, the optic thalamus, the crura cerebri, and the medulla oblongata. Lussana located vasomotor fibres in the optic thalami, and Christiani ascertained that removal of the hemispheres caused fever. Aronsohn and Sachs have, however, shown that the vasomotor fibres are not the cause of the increased temperature, that removal of the hemispheres alone will not cause fever, and that there is no increased production of heat in the muscles nor any marked vasomotor changes. At the present time, therefore, the only heat-centre that has been localized is that at the anterior and inner portion of the optic thalami in rabbits, and probably in an analogous

location in the human being. It has long been known clinically that some lesions of the spinal cord are capable of causing considerable increase of temperature, but exactly how this is produced is not known. Eulenberg and Landois have also shown that excitation of one cerebral lobe can produce a notable elevation of temperature in the opposite limb, although they do not speak of the general temperature. Richet has performed a number of experiments upon rabbits and dogs, showing that the excitation of different convolutions is capable of causing a general thermic rise. Thus injury to the anterior lobes caused an elevation of temperature of 3.6° C. in forty-five minutes, whilst cauterization produced the same phenomenon. The method of production of this increase of temperature is largely a matter of speculation.

CHAPTER II.

ELECTRICITY.

It is not my purpose to do more than deal with the applications of electricity in nervous and mental disease. I am not writing a text-book on electricity, and I shall therefore not deal with theoretical considerations, except so far as may occasionally be necessary in order to banish the confusion existing in the average medical mind about electrical terms and still more about electric phenomena. If by some magic power I could at this very moment unroof the office of every physician in the United States, I hesitate to mention what I think would be the proportion of worthless batteries, or worthlessly used batteries, or worthless rheostats, or worthless milliampère-meters, or electrodes entirely unfitted for the purpose to which they are daily being put. I believe, therefore, from experience as a teacher and consultant, that a brief space can be advantageously devoted to the instruments with which electricity is used and the methods of handling them.

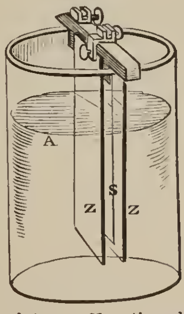
Electricity, as we use it in medicine, is of three kinds:

The *Galvanic*, which is sometimes called the voltaic or the continued current.

The *Faradic*, which is sometimes called the faradaic, or the interrupted.

The *Static*, which is made by friction, generally in the Holtz or similar machines.

FIG. 64.



A cell. Z. Positive plate. S. Negative plate. A. The fluid.

Galvanic electricity is a force produced by the immersion of two dissimilar metals in a fluid capable of inducing chemical action. The two metals, or *plates*, immersed in a fluid, and the jar which holds them, are called collectively a *cell* (Fig. 64). A collection of cells constitutes a *battery*. One of these metals, or plates, is called

the *positive*, and the other the *negative*. The current flowing from the positive plate is called the positive current, and that from the negative plate is termed the negative current. The two plates differ in their chemical and physiological properties. We may assume for practical purposes that the metal which is the most actively attacked by the liquid is the positive metal. All metals, therefore, can be ranged in a comparative series from the most positive to the most negative, each one being positive or negative to other members of the series. Within the cell the current flows from the positive plate Z, to the negative plate S, Fig. 64. If to each one of these plates is attached a wire, and the wires are joined, the current flows through them and back again into the cell, but in the opposite direction to the current in the cell. These wires are called the *poles*. If between the two poles a human body is so interposed that the current must flow through it, the direction of the current will be unchanged. The distinction that is here described between the plates in the cell and the wires outside the cell should be fixed clearly in the mind of the reader, as it is a matter about which there is much confusion. The positive pole is known as the anode, and the negative pole as the cathode.

Cells vary in construction according to the uses to which they are to be put. If a wire is simply to be brought to a white heat, as in the electric cautery, the cell should be constructed in an entirely different manner from what is required when the wire is simply intended to pass a current of electricity through the human body. When a galvano-caustic is needed, the largest possible amount of electricity should come from the cell, as, the flow of the current being through a metallic wire which conducts electricity well, the resistance opposed to the current will be very slight, and therefore the power that the current has of overcoming the resistance, or the electro-motive force,[1] as it is technically called, need only be very slight. If, on the other hand, a moderate quantity of electricity is to pass out of the cell and push its way through the human body with its enormous resistance, the quantity of the current is unimportant in comparison with its capacity for overcoming resistance, and the electro-motive force must accordingly be great. In other words, the quantity of the current and its capacity for overcoming resistance, or its electro-motive force, are two entirely different things, and for this reason the battery which is used for electric cauterization cannot be used for electrification of the human body. It is of importance

[1] The electro-motive force is the result of the difference of electric potential of two bodies, or two different parts of the same body, and it is proportional to this difference. It is often compared—and justly—to the difference in pressure of the water in a water-pipe at two different levels. Practical electricians, however, have come to use the term as synonymous with its power of overcoming resistance; and although this idea is not strictly accurate, I have found that it conveys a more definite idea to the average mind, because the whole doctrine of potentials is confusing from its very arbitrariness. The zero potential, for example, is assumed to be the potential of a spot infinitely distant from all electrified bodies, and the earth is dogmatically asserted to be this zero potential. Anything positively electrified is asserted to be at a higher potential than the earth, whilst anything negatively electrified is at a lower potential than the earth. If the difference of potentials between two bodies connected with each other is maintained by the friction of a Holtz machine, or the chemical action of a galvanic battery, an electric current results, and the difference between the potentials of these two bodies determines the electro-motive force. But the difference between these two potentials, producing the electro-motive force, is to all intents and purposes the capacity of overcoming resistance.

for the physician to know what cell is best adapted to the exigencies of practice, what cell is most easily carried about, lasts the longest, gives the most constant current from day to day, and is the cheapest. All these details will be considered later. For example, the telegrapher makes use of a Daniell's cell that is large and cumbrous, and that has to be replenished every day or two; and as his instrument is never moved, and as it is very easy for him to replenish one or two cells, which are all that he needs, the Daniell's cell answers his purpose very well. But it would be entirely too large and require entirely too much attention for the physician, more especially as a number of them would be needed by the latter to furnish the requisite current. Then, again, the stationary cell, which the physician uses only in his office, may be much larger and more cumbrous than those which he has to carry around with him in his carriage to the bedsides of his patients.

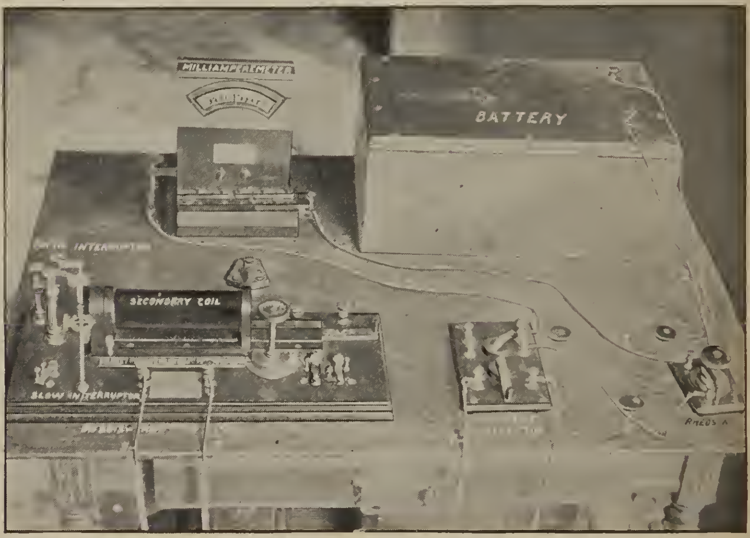
Owing to its low resistance, its flexibility, and its cheapness, copper is the metal generally used in the wires or poles. If brought directly in contact with the naked human body, these wires would be found to be painful, since they concentrate the current upon too small an area of skin. To prevent this pain and to diffuse the current over a wider surface of tissue the poles have attached to them a diffuser or electrode, a device varying in size, shape, and material, according to its purpose. If, for example, the current is to be concentrated upon a small nerve, a small piece of copper is preferred. If the current is to be diffused over a large area, the electrode may be such as shown in Fig. 67. Electrodes are usually constructed of copper, covered with sponge or absorbent cotton in order still further to diminish the painfulness of the naked copper when placed upon the textures of the body. In some cases they are made of clay.

It is necessary to have some accurate means of measuring the current, and for this purpose an instrument known as the *milliampère-meter* has been devised. The ampère is the unit that has been adopted of late years for the quantity of electricity, and the milliampère, or thousandth part of the ampère, is the unit of quantity which is best adapted to the uses of the medical electrician, since we scarcely ever use the full ampère, but rather proportion our doses of electricity in quantities that range from one to ten or twenty milliampères in medico-electric therapeutics, and from three hundred to five hundred milliampères in electrolysis. The different kinds of milliampère-meters will be considered in their proper place. Until recently the importance of accurate dosage of electricity has not been appreciated. To follow the old custom of measuring the current by the sensations of the patient is no more rational or scientific than to direct a patient to take as much quinine as he thought would produce cinchonism. Another equally faulty method has been to use a given number of cells for a case, but this is grossly inaccurate, since the resistance of the human body varies enormously under varying circumstances, and the current developed from the cells is likewise subject to much fluctuation.

It is also necessary to have a regulator of the current, so that we

can admit as much electricity as we want. Such an instrument is called a *rheostat.* The old fashion was to turn on a sufficient number of cells, and then, if that did not give enough current, to turn on another cell, and so on until the requisite amount of current was admitted. The difficulty with this method should be very apparent. Suppose that we turn on five cells, and then we find that we have two milliampères of electricity measured off; but we want three milliampères. We therefore turn on another cell, but we find that this gives us four milliampères. Now, what are we going to do? If we turn back to three cells, we will only have two milliampères; if we turn on four cells, we have four milliampères. If we have something in the circuit that will accurately regulate the admission of the current, so that we can turn on just three milliampères, it is very

Fig. 65.



Battery table showing arrangement of galvanic and faradic apparatus, rheostat, and milliampèremeter.

apparent that we are very much more precise. Besides this, it may be very unpleasant to turn on each additional cell, for in doing so we give the patient a shock. If we were passing electricity only through the human trunk, the shock caused by adding a whole cell would be very slight, and the patient would not complain much; but if we were passing electricity through the human brain, a single additional cell would give a shock so unpleasant as to cause vertigo, a flash of light, and often considerable distress. It is therefore further apparent that a regulator that will turn on or turn off the current very gently, without shock, is eminently desirable, and such a regulator is the so-called rheostat. The different varieties of rheostats will also be considered hereafter.

Let us now suppose that we have a patient to whom we want to give galvanic electricity, and that we desire to make use of the cell with its wires or poles attached, these wires being armed with the proper electrodes, and that we also wish to make use of a rheostat or regulator of the current, and of a measurer of the current, or milliampèremeter. Fig. 65 shows how this is done. To the battery are attached the two poles or wires, 1 and 2. Disregarding wire 1 for a moment, we turn our attention to the other pole or wire (2) going to the rheostat, thus conducting the current into and through it. On the other side of the rheostat the current is carried off through another wire, 3. This runs to the milliampèremeter, conducting the current into and through it. From the other side of the milliampèremeter the current is taken by wire 4. The wires 2, 3, and 4, with the intervening rheostat and milliampèremeter, therefore constitute one pole of the battery just as the wire 2 does before it has been connected with the rheostat and the milliampèremeter. If we put an electrode on wire 4, and another electrode on the other wire, 1, which constitutes the other pole of the battery, and if between these two electrodes we put the human body, the current flows through the body and around again into the cell of the battery. The current-collector shown in Fig. 65 has the wires attached to it, and has a simple switch which can be turned to either binding-post, and thus enables us to direct the positive or the negative current into either one of the wires 6 and 7.

THE FARADIC, OR FARADAIC, OR INDUCTION CURRENT.

The faradic current differs considerably from the so-called galvanic current. The latter, as we have seen, is a current that flows out of a battery through the human body back again into the battery, not being interrupted in its course in any way. In the galvanic battery we usually make use of several cells, varying in number from five up to fifty, or even a larger number if electrolysis is to be effected. Galvanic electricity, therefore, may be defined, for our present purposes, as a *continuously flowing current of large quantity, with sufficient electro-motive force to enable it to overcome the resistance of the human body;* and its therapeutic effects depend mainly upon the quantity of the current which is endowed with sufficient power of overcoming resistance, or electro-motive force, to enable it to penetrate the body. Faradic electricity, on the other hand, is the current which flows from one or two cells, but which, by means of various mechanical devices, is interrupted constantly, and made to have more electro-motive force or much more power of overcoming resistance than galvanism. The general principle underlying this phenomenon we will now endeavor to demonstrate. Suppose that there is a cell to the plates of which are attached the usual poles or wires. Let us join these poles or wires so that the current will run around them back into the cell. Let us now place near these wires another wire, and in the latter we intercalate a milliampèremeter, so that we can detect any current of electricity that shall pass through this wire.

At the moment when the current passes through the wires attached to the cell we shall see that the needle of the milliampèremeter intercalated in the wire fluctuates, indicating that a current of electricity is passing through this wire. Yet the wires do not touch, and it is evident that while the current in the milliampère wire is due to the passage of the current in the battery wires, it is not due to the contact of the two wires. It must therefore be by *induction* without contact. A current is *induced* in one wire by the passage of a current through a neighboring wire. The battery wire is called the primary wire, and the milliampère wire is called the secondary wire. In this secondary wire the milliampèremeter is intercalated. It is a curious fact that the current in the secondary wire is not observed while the current is quietly passing through the primary wire, but only at the moment when the current is suddenly turned on in the primary wire, or suddenly turned off in the same. Let us suppose, therefore, that in the battery wire we have a mechanical contrivance

FIG. 66.

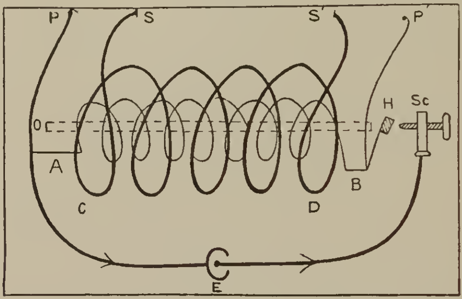


Diagram illustrating the primary and secondary wire of a faradic battery. (From DE WATTEVILLE.)

for turning on and turning off a current, for making or breaking the current, as it is technically called, or interrupting it. It is evident that such an interruption will be a great convenience in producing a current in the secondary wire. This principle of induction and interruption constitutes the keynote of the faradic battery, and hence we can understand why it is called the interrupted current, in contradistinction to the steadily flowing current of the galvanic battery. In every faradic battery, therefore, there is a device for interruption, and also a primary wire which carries the current and which induces the secondary current in the secondary wire. This primary wire is usually in the shape of a long coil wound around a bobbin, and over it or in it is another long coil holding the secondary wire, also wound around a bobbin. Fig. 66, taken from De Watteville, illustrates this. This shows the primary wire, A B, one end of which is connected to the battery, E, and the other end is connected to the hammer, H. Inside the coil of the primary wire is a bundle of iron rods, O, and C D is the secondary coil, which is entirely independent of the rest of the apparatus. The current from the primary wire

can be collected from the two poles, P and P′, and the current from the secondary wire can be collected from the two poles, S and S′, so that either the primary or the secondary coil can be used. When the current flows in the primary coil, A B, the iron, O, becomes a magnet by the passage of the electricity through it and the hammer, H, is immediately attracted by this magnetic action, and is drawn away from the screw, SC. Then the current ceases flowing. When the current ceases to flow, however, the iron ceases to be a magnet, and ceases to attract the hammer magnetically, and this hammer flies back, by means of the elastic spring, to the screw, SC. Then again the current from the primary wire flows around, and the same process is repeated. The distance of the hammer, H, from the screw, SC, will determine the rapidity of the interruptions, and therefore every apparatus has a means of regulating the swing of the hammer, so that the interruption may be quick or may be slow.

It is thus apparent, as we have said, that the faradic or induction current is a current of small quantity, which, by means of interruptions, is made to produce another current by induction. The primary wire, therefore, of a faradic battery would conduct exactly the same electricity as the wires of a galvanic battery if it were not for the interruption; but the secondary wire of a faradic battery not only differs from the galvanic current by means of the interruption, but also because of the fact that it is an induced current and does not flow directly from the cell. It is, therefore, evident, as we have said, that the faradic battery is an interrupted current, and also an induced current, and the result of this interruption and induction is that its electro-motive force, or capacity for overcoming resistance, is increased. The galvanic current, on the other hand, is a continuously flowing current. As experience has shown that one or two cells are usually quite sufficient to give the requisite current to a faradic battery, whilst it has also been demonstrated that from twenty to three hundred cells are necessary to give the requisite current for a galvanic apparatus, there is the further distinction that faradic electricity is a small amount of electricity made more penetrating, or endowed with a higher electro-motive force, by the mechanical devices which have been described; whilst the galvanic current is a current of large quantity with just sufficient electro-motive force, or power of overcoming resistance, to enable it to penetrate the human body.

The wires, or poles, and the electrodes are the same for the faradic battery as they are for the galvanic battery. The measuring-apparatus and the regulating-apparatus of a faradic battery are, however, very different from those of a galvanic battery. Galvanism, as we have seen, can be measured by a milliampèremeter and regulated by a rheostat. In the faradic battery, however, there is no precise means of measuring the current. The galvanometers that are necessary to this form of current are very expensive, and only indicate the quantity of the current, but they do not indicate the suddenness of the interruption, upon which, as we have seen, depends the flow of the current in the primary and the secondary wires. In measuring faradism, therefore, there is usually a scale along which the outer

bobbin containing one of the wires is pushed, and this enables us to say that to-day at a certain point we have produced such an effect, and that to-morrow we shall probably produce the same effect when the bobbin is pushed to the same point. This measurement is, as must be apparent, dependent upon the assumption that the cell and the resistance of the body shall not vary daily, and we know as a matter of fact that cells and the resistance of the human body do not remain the same from day to day. Practically, therefore, we can only speak of a faradic current as moderate, strong, or painful, determining this by the sensations of the patient and by the amount of muscular contraction that is obtained by applying the current to a muscle or to the nerve attached to a muscle.

STATIC ELECTRICITY.

Static electricity is manufactured for medical purposes by means of the Holtz machine, which, as I scarcely need to say, consists of two upright jars—so-called Leyden jars—and a revolving wheel fixed in front of a stationary plate of glass. This revolving wheel is made to revolve by means of a handle affixed to the same wheel, to which is attached a rubber cord encircling a revolving axle, on which the wheel is fixed. As this wheel revolves it brushes against several small wire brushes, and the electricity is generated by means of this friction and flows off on to the metal combs, which conduct it to the Leyden jars. From these it flows on to the handles above the Leyden jars, and to these handles is affixed a metal chain armed with the different electrodes, consisting of glass rods tipped with metal cones; so that the electricity passes off in the form of electric air, or electricity diffused through the atmosphere, or in the form of sparks. When it is desired to obtain the electric air, the handles are separated widely so that a spark cannot pass across from the bulbs on the handles. If the sparks are to be carried directly into the body of the patient, the bulbs on the handles are approximated, and the length of the spark passing into the human body will be approximately proportionate to the length of the spark between the bulbs.

GALVANIC BATTERIES.

In selecting a battery a physician should always be assured that the cells composing the battery will afford a constant current—in other words, a current which will not vary greatly from day to day, and which shall not need frequent refilling of the cell. It is not too much nowadays to require of a cell that it shall run at least eighteen months to two years without replenishing; and I have frequently had cells that have so run from three to five years, although I have never had one that lasted over two years if it was in constant daily use. If it is intended to use the battery stationary in the house, office, or hospital, the size and weight of the cell will be a matter of little consequence, and attention should only be paid to the con-

stancy. If, on the other hand, it is desired to use the battery as a portable one, so that it can be carried around to the houses of patients, the size of the cell becomes of very great importance. The physician must, therefore, make his selection among the cells that I am now about to speak of with regard to these requisites

The different cells which are now used in this country in making galvanic batteries are: the Grenet, the Leclanché, the chloride of silver, the Stammers, the Axo-Leclanché, the Law, and the Edison-Lalande.

The Grenet cell is a fairly good one as long as it lasts, but unfortunately it needs to be replenished every few weeks, and this is a matter of some little inconvenience. Moreover, as it is an open cell and contains fluid, it is liable to spill, and although the battery-makers maintain that this can be prevented by what is called a hydrostat, or a board covered with padded rubber, which is slid in over the top of the cells and pressed down by a rod from the lid of the battery, yet it will be found that leakage will occur as the rubber grows old and shrinks, whilst the rubber is apt to adhere if left in place over the cells for any length of time.

The Leclanché is made as a liquid, and also as a dry cell. The liquid cell gives a fairly constant current as long as it lasts, and it will run from six months to a year, when it will usually have to be refilled. This refilling process is simple, consisting only of pouring in some sal ammoniac in solution, but, nevertheless, it consumes some little time. The size of the liquid cell militates against its being used as a portable battery, in view of the equally, if not more, efficient portable cells that are now to be had. The dry Leclanché cell is certainly an excellent one; and it is claimed that, with proper care, it will run for about eighteen months. I have run one for thirteen months. The small weight of the cell certainly renders it invaluable for portable batteries.

The chloride of silver is an admirable cell for galvanic purposes, if properly constructed. The one used in this country is the Barrett chloride of silver cell, and was first made for me some thirteen years ago by Mr. John A. Barrett. These earlier ones were extremely durable, for a battery in my possession lasted practically unimpaired for three years, although in daily use, and was finally broken by a fall; and I know of one now in this city (New York) that has lasted for five years, although I am not aware how much use it has had. Since this time, however, it has sometimes been very poorly, and sometimes very well made. In every case in which it is used it is only proper that some guaranty from the makers should be had of its durability. It is the most portable of all the cells, each one weighing only six ounces.

The Stammers cell has the advantage of being a very cheap one, as it costs only twenty cents. The maker claims that it will run eighteen months. I have never used it, and cannot, therefore, say anything of my own personal experience.

The Law cell is very constant and requires scarcely any attention. I have had a battery made of it in daily use in our large clinics at

the New York Polyclinic for two years. Its size, however, will prevent its use in portable batteries, although I should prefer it, for my own part, to any other cell for a stationary battery.

The Edison-Lalande is a new cell, and, as the name indicates, is a modification of the Lalande cell by Thomas A. Edison. It is claimed that it is practically indestructible—that is, it will run as long as it is not absolutely worn out, and this is about a year. I have had no experience with it, but I should certainly think it worthy of a trial in stationary batteries, although its size will not allow it to compete with the smaller cells for portable batteries.

The liquid Leclanché cell is used in this country in both stationary and portable batteries, although its weight makes it unfit for the latter. The dry Leclanché cell is used in some portable batteries. An accessory battery is also made, containing any number of cells that may be required, and that can be attached to the main battery at any time when more current is required, as for purposes of electrolysis. These are extremely useful.

Each one of these batteries has its own devices for collecting the current at certain binding-posts, to which wires or rheophores can be attached. Great attention should be paid to these binding-posts. It is customary to make them in such a manner that the wire conducting the current to the patient's body passes either into a split post which holds it by its elasticity, or else passes into a hole and has a screw to screw down upon it. The split binding-post should be entirely discarded, for it loses its elasticity with use, and the wire is apt to slip and give unpleasant shocks to the patient. The other binding-posts, perforated with a hole through which the wire is passed, and with a screw which screws down upon the wire, is always the best, and it should be carefully ascertained that the screw works upon a strong thread and holds well, as many batteries are defective in this detail. Kidder makes a spring binding-post which is said to be excellent. In some of these batteries there is a current-selector, whose purpose it is to take into the circuit as many cells as may be desired. This ought to be entirely discarded, as the current-regulator, or rheostat, is a far better instrument. When this rheostat is used, all the current from all the cells can be collected directly from the binding-posts, and the amount of current that is admitted into the patient is thus regulated by means of the rheostat, and this can be done gradually and without shock, whilst the current-selector invariably causes a slight shock as the handle passes from cell to cell. An attempt has been made to obviate this latter defect of the current-selector by having the handle make connection with one cell before it leaves the other; but this does not answer, as can readily be demonstrated when the current is passing through the human brain, for however carefully and gently this collection may be made, the patient will see sparks or have a slight sensation of dizziness.

FARADIC BATTERIES.

There is no great difference in the efficiency of the different faradic batteries upon the American market, with the possible exception of the chloride of silver cell, which has not worked well in my hands in the faradic apparatus, being quickly exhausted. The only criticisms that I have to make of most of the faradic batteries are that generally the secondary coil is too short to give sufficient intensity of current when it is intended to affect such deep-lying structures as the abdominal organs, and that the long interrupting apparatus is apt to be defective. As we seldom need very intense currents, however, and as the long interruption is a therapeutic measure of infrequent application, these two defects are not usually of much importance. In a stationary apparatus, however, I should always make use of the Dubois-Reymond coil, which has a long interrupter of excellent workmanship, and in which the secondary coil is adequate for all purposes. This is represented in Fig. 65.

STATIONARY BATTERIES.

In having a stationary battery constructed I think that the physician will usually do best with the Law, the wet Leclanché, or the Edison-Lalande cell, and of these I should give the preference to the Law. The cells of the stationary battery may either be in a closet in the physician's office, in the kitchen, the cellar, or a so-called cabinet. In all cases, however, the connecting wires leading from the battery to the rheostat and milliampèremeter should be easily accessible, so that any defect in the conduction may be readily remedied by the physician himself. The connection between the cells of the battery itself will usually require the attention of an electrical mechanic if conduction becomes impaired. In my own office I have not made use of a cabinet because of this desire to have the connections, so far as feasible, immediately under my own eye. I therefore place the rheostat, milliampèremeter, binding-posts, and Dubois-Reymond coil upon an ordinary flat table, which can be purchased at any furniture store. (Fig. 65.) This table can be covered, if desired, with a glass cover to protect it from the dust and from meddling.

In some batteries there is a device for sending the galvanic and faradic current through the same wires. This is apt to get out of order, however, and is of no practical value whatever, as it is no therapeutic gain, but rather a disadvantage, to give the galvanic and faradic currents simultaneously. I had one in my office for a number of years, but finally abandoned it.

It is an easy matter for the physician to find out where the fault lies if the current does not pass well. Fig. 65 shows how this can be done. It is evident that the defect must lie either in the battery supplying the electricity, or else in some of the conducting apparatus between the battery and the patient. Let us, therefore, go over each

segment outside the battery. First one electrode should be removed, and the tip of the wire from which it has been removed applied to the other electrode. If the current now passes, it is evident that the fault has been in the electrode that has been removed. If the current still does not pass, remove the next electrode and touch the tips of the two wires together, and, if the current passes, the fault must have been in this electrode just removed; or if it does not pass, then remove wire 7 and put a reliable one in its place; and in this manner wire 6 should be tested, then wire 5, then the rheostat, and, finally, in succession, wire 4, the milliampèremeter, and wires 3, 2, and 1. The defect will be found to be in one of the segments removed, or it must be in the binding-posts, or the wires leading from the binding-posts to the cells of the battery, or the cells themselves.

It should be borne in mind that the more delicate of these cells that have been described, such as the chloride of silver cell and the Leclanché, are destroyed by what is called "short-circuiting." If the current be passed around from the cells through the different wires of Fig. 65, and back again to the cells without the interposition of some body between the two wires whose resistance is equal to that within the body of the cell, this will be what is technically known as the "short circuit," and it will paralyze the delicate cells. For this reason the tips of the wires or the electrodes should never be allowed to come into contact when the current is turned on, unless there is a rheostat in the circuit; and a still better method is always to keep the pole-changers on the galvanic apparatus away from the binding-posts leading to the battery. In the faradic batteries this short circuiting need not be taken into account.

MILLIAMPÈREMETERS AND RHEOSTATS.

Until within a very short time a good milliampèremeter was not obtainable in this country, and I believe that the first reliable one was made for me some twelve years ago. In purchasing one there are certain requisites to which the physician should carefully look. In the first place, the scale should be graduated according to the needs of the particular person using it. If electrolysis alone is to be employed, the scale should run up to four or five hundred milliampères and over, and it is of small importance whether a fraction of a milliampère can be measured off. If galvanism of the spinal cord and the brain alone is to be done, the scale that mounts to thirty milliampères will be quite sufficient; but it should be distinct enough to indicate a half or a quarter of a milliampère. If both spinal and cerebral galvanization and electrolysis are to be done, there should be a combination of these two scales, so that it will be possible, on the one hand, to measure off a fraction of a milliampère, and, on the other, to obtain four or five hundred. In the second place the needle should move with very little oscillation, so that, after marking off the requisite amount of electricity, it shall either not swing at all or come to a stand in a second or two. In this particular most of the instruments are

exceedingly defective. In the third place, the milliampèremeter should always have an upright scale, numbered with sufficient distinctness to be easily read at a distance of several feet, or otherwise it will be exceedingly awkward to employ it. Most milliampèremeters in this country are not graded by one general standard, so that when one milliampèremeter measures one milliampère, another may measure two or three; indeed, in a trial about two year ago in the office of Dr. A. H. Goelet, of this city, there were found to be startling variations between the different instruments, although since this time the instrument-makers claim that the instruments are almost alike. Still another requisite of a milliampèremeter is that it should be portable. As best combining these requisites I unhesitatingly recommend the Vetter milliampèremeter. The one which is pictured in Fig. 65 was made by Hirschmann, of Berlin, and it is a very accurate instrument which has stood years of wear; but it is too cumbrous to be portable, and the needle swings exceedingly. Edelmann, an instrument-maker of Germany, also makes an excellent instrument which can be purchased in this country.

The rheostats in use at the present day for physicians are either the water or the carbon ones. The objection to the former is that chemical changes take place in the water, so that after an instrument has been in use for awhile unpleasant shocks are apt to be given, and some accidents have occurred in this way. The new Bailey rheostat is the best of all the water-rheostats, and it is claimed that all previous defects have been overcome in it. The best rheostat made in this country, however, is the carbon instrument made by Vetter, which is shown in Fig. 65. I have an instrument by which I can turn on $\frac{1}{8}$ of a milliampère with perfect ease and without the slightest shock. Vetter and Stammers also have rheostats, or converters, which can be affixed to the ordinary Edison light in such a manner as to control the current and adapt it to purposes of spinal and cerebral galvanization. I have given up the use of the Edison current, however, for cerebral galvanization, as exceedingly unpleasant though slight shocks are frequent, probably due to variations in the current; moreover, the quality of the current is apt to be unpleasant. But I find it unobjectionable when employed about the trunk or the limbs, except when the nerves are sensitive, as in some cases of neuralgia and neuritis. For running the galvanic apparatus, however, these converters are exceedingly convenient, as, indeed, they are for galvanic purposes. For purposes of electrolysis, however, and for electric lighting, the Edison and alternating currents can be made approximately safe by a proper rheostat.

WIRES AND ELECTRODE OF GALVANIC AND FARADIC BATTERIES.

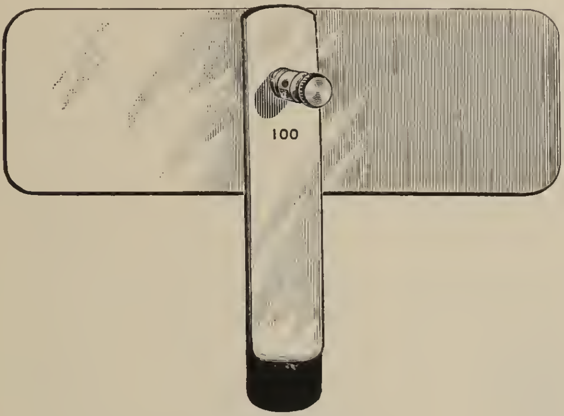
The wires, or rheophores—the latter term being very seldom used by American electricians—should always be made of tinsel cord woven of a large number of well-made tinsel threads, as this has

been proven to be the best conducting material for medical batteries. This tinsel thread is always covered with silk as an insulator of the current, and it does so insulate as long as the silk is not wet. As we shall have occasion to see, however, water must be freely used in the administration of electricity for medical purposes, whereby the silk thread is very apt to become soaked and thus allow the electricity to pass to the body of the patient when the thread accidentally comes in contact with the skin, so that slight, unexpected, and annoying shocks will be given in this way and unnecessarily frighten timid patients and children. To obviate this the wires should be run through little rubber tubings (which can be obtained of all the instrument-makers), and thus be completely insulated. These wires should always be of a generous length, so as to enable them to reach out a good distance from the patient and facilitate applications, more especially when the patient lies upon a couch or lounge. I have mine always made at least two and three-quarters yards in length, as almost all the companies supply wires of very defective length. At one time I made use of copper wires, but I found that they were not only brittle and awkward to handle, but experiment has since demonstrated that they are really not so good conductors as tinsel wires woven of many threads of perfect tinsel.

The size of the electrode is a matter of very considerable moment. The object always is to get a certain amount of electricity into the body of the patient and to impinge it upon the organ that it is desired to affect. There are certain peculiarities of the distribution ot the electric current that should be borne in mind in attempting to effect this purpose. If two electrodes, each being two inches in diameter, are placed, for example, upon the spinal cord, and a current passed, a certain amount of the current will pass in an approximately straight line directly from electrode to electrode, whilst a certain other portion of the current will be diffused in curvilinear lines to each side of this straight line. If the same amount of current is passed from two electrodes placed exactly in the same places, but these electrodes are made four inches in diameter instead of two, more of the current will be diffused in curvilinear lines and less will pass directly in the straight line. If, therefore, we wish to reach the spinal cord lying deep beneath the muscles of the back, the vertebræ, the loose areolar tissue of the spinal canal, and the membranes of the cord, we must do it by the diffuse current as represented in these curvilinear lines; and therefore the greater the size of the electrodes and the consequent greater diffusion of our current, the more it will affect the spinal cord. For this reason the small electrodes which are usually sold with batteries are absolutely worthless for purposes of spinal applications, and in their place should be used as large electrodes as practicable, so that in the adult the electrode of Fig. 67 should be employed, and in the child the electrode of Fig. 68 or 69. If, on the other hand, it is desired to have the current impinge upon a nerve-trunk, it must not be too diffused. It will, therefore, be best to make use of a large electrode (Figs. 67 to 71) for the one pole which is not to be placed upon the nerve, and one of the smaller

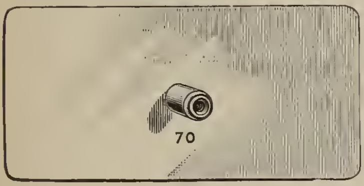
electrodes, such as the electrode of Figs. 72 to 76, for the other pole which is to be put directly upon the nerve. There is a limit, how-

FIG. 67.



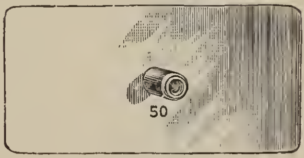
18 x 6 centimetres.

FIG. 68.



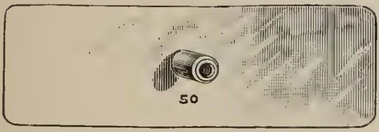
18 x 15 centimetres.

FIG. 69.



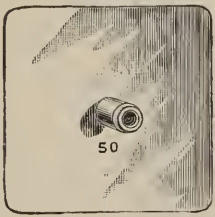
10 x 5 centimetres.

FIG. 70.



5 x 4 centimetres.

FIG. 71.



7.3 x 7.3 centimetres.

FIG. 72.

3 cm. square.

FIG. 73.

5 cm. square.

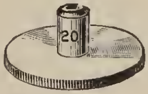
FIG. 74.

13 cm. square.

FIG. 75.

15 cm. square.

FIG 76.



20 cm. square.

ever to the diminution in size of the latter, because if it is too small it will cause pain, and practically the smaller electrode should be

made use of for this purpose, and not the smallest one, Fig. 72. The large electrode that has been mentioned admits the current more diffusely, whilst the smaller one causes it to impinge, in entering the body, upon the particular nerve that it is desired to affect. If both the electrodes are as small as the electrode of Fig. 72, it will be found that the administration of the current will be more painful. The electrodes must, therefore, be made of different sizes and also of different shapes, according to the purposes for which we desire to use

Fig. 77.

Fig. 78.

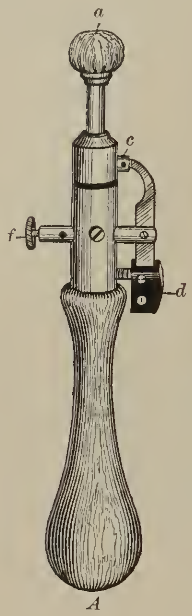


Fig. 79.

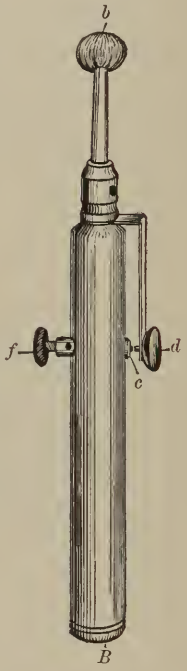
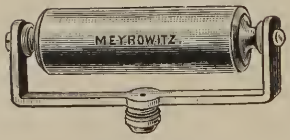


Fig. 80.



Roller electrode.

them. Erb has suggested that it would be well to have electrodes of definite sizes, so that electricians throughout the world could compare results with more approach to precision, and he has therefore figured the different sizes and given them numbers. These are represented in Figs. 67 to 76. They are made of malleable copper, which should be of sufficient thickness to bend without breaking

and to preserve its shape after being bent. Most of them are very defective in this respect. They should be covered with absorbent cotton, taking care to pad this carefully around the edges and the angles, so that the electricity may not cause smarting at these points, which it is very apt to do when the electrode is bent around the neck or a limb. The cotton can be held in place by muslin or linen stitched on, which can be removed in a moment and is a great advantage in point of cleanliness. Most manufacturers cover their electrodes with a fine sponge, glued on. This is a filthy contrivance, and ought to be entirely discarded, in view of the fact that we have so cleanly, so easily affixed, and so cheap a cover as the absorbent cotton. For applying electricity to the pelvic organs of the male, I have had made an electrode which is six inches wide and thirteen inches long. It consists of a malleable copper plate, below which is attached a plate of rubber projecting well beyond the edges of the copper and serving as a protection against the water which must drain off. Between the plate and the rubber is placed a thick mass of absorbent cotton, held in place by muslin or linen stitched on as has been described. This electrode is placed upon a stool, the patient sits down upon it in such a manner that the perineum comes in contact with the cotton, which should be thick and abundant, and thus the electricity is conducted well up into the perineum. Dr. A. H. Goelet, of New York, has devised a clay electrode, which is valuable in uterine and vaginal applications.

The ordinary uterine, rectal, urethral, nasal, and aural electrodes are too well known to need reproduction here.

Wetting the skin with water is the simplest method of diminishing the resistance offered by the skin, and this water should be as warm as can be borne conveniently according to the season of the year. Water does not reduce the cutaneous resistance quite as much as acidulated solutions, yet there is no practical difference. Every physician using a battery much in his office should therefore have upon his table a large jar to hold this water, which should be changed for each patient, and in it should be a large sponge with which to wet the skin; or a wash-basin with running hot and cold water should be at hand. The cotton or sponge coverings of the electrodes must also be wet, as they are very poor conductors when dry. Great attention should be paid to this wetting of the skin and electrodes, and I insist upon this, as I have again and again heard complaints about the feeble currents of batteries, and have found the real reason to be that neither the skin of the patients nor the electrodes were thoroughly wet.

The resistance of the human skin varies very considerably in different places and somewhat under different circumstances. The most conclusive researches on this subject have been undertaken by Jolly, of Strassburg. He has shown that, contrary to what is generally supposed, the inner surface of the hands and the soles of the feet offer but a relatively slight resistance; yet on this inner surface of the hands, which were examined in ten males, the

resistance varied between 22,000 and 55,000 Siemens units,[1] and in ten females between 16,000 and 50,000 Siemens units, whilst in the feet the variations were in males between 15,000 and 32,000, and in females between 20,000 and 55,000 Siemens units. In the face, upon the skin of the cheeks, the average resistance was 43,300 Siemens units in males, and in females 78,000, whilst the temporal region of the face was on the average 92,500 in males, and in females 109,000. For all other areas of the skin the average resistance was over 100,000 Siemens units, generally between 200,000 and 300,000 Siemens units. These facts concerning the resistance of the human skin have been marvellously little known, and it is an actual matter of evidence that when killing by electricity was first recommended in New York a few years ago, the commission reported a method based upon the assumption that the average resistance of the human skin was only 2400 ohms! These cutaneous resistances vary enormously. The following average resistance of the different regions is given by Jolly, in Siemens units :

Soles of the feet	23,000
Palms of the hands	41,300
Cheeks	42,500
Temporal region	92,500
Neck	21,900
Dorsal surface of the feet	236,000
Thigh	275,000
Back	289,000
Dorsal surface of the hands	304,000
Leg	324,000
Arm	355,000
Forearm	364,000

The resistance of the skin is enormously reduced by the passage of a galvanic current. Thus, in one case of Jolly's where the resistance in the forearm was 113,700 Siemens units before the passage of the current, it was reduced in five seconds by the passage of a current from twelve Stöhrers cells to 52,000 Siemens units. But this resistance to the passage of the current varies in different cutaneous areas, so that, for example, the resistance of the skin of the inner surface of the hands and the soles of the feet will not be very much reduced by the passage of the current. As I have already said, moistening the skin will also reduce the resistance, and the warmer the water the more the resistance will be reduced. Saponification of the water will answer still better, and also acidulation of it, and many of the text-books recommend this latter method ; but anyone who has had any practical experience in giving electricity will know that this consumes much time and tarnishes the metallic fittings of the electrodes. The resistance of the skin will also vary in different persons with different states of the weather and with different diseases.

It is a very convenient matter to have what is called an inter-

[1] The Paris or legal ohm is the usual American and British unit for measuring resistance, and is arbitrarily understood among electricians to be a standard coil of copper wire one square millimetre in diameter and about 48.5 millimetres in length, attached to a freshly charged Daniell cell, or a column of mercury one square millimetre in area, 106 centimetres in height, at a temperature of 0° C. The Siemens unit would be represented by the same column of mercury with a height of 1 metre ; so that one ohm is equal to 1.0600 Siemens units. It has recently been proposed to have still another unit of resistance, to be called the Kolvin, after Colvin, the electrician, but this has not yet come into use.

rupting electrode; that is, one in which the current can be turned off or on with a mere pressure of the finger. If, for example, we are giving electricity to a patient, and it is necessary to turn off the current, or to turn it on, or to break and make the circuit, as it is technically called, this can be done by taking the electrode away from the skin, and thus breaking the current, and putting it back on the skin, and thus making it. But the disadvantage of this method is that a fine nerve, which it may be very difficult to locate, such as the popliteal in fat people, may slip from under the finger. Some device, therefore, that will break the current without taking away the electrode from the skin, is advisable, and this is simply done in the so-called interrupting electrode. The best one of these is that figured in Fig. 79, which I have had made for myself, and in which the circuit is made by pressing down the little trigger, and thus allowing the current to pass, whilst taking the finger away from the trigger breaks the current The trigger in this electrode works as easily as the hair-spring of a revolver, and the mere tap of the finger is sufficient. Fig. 78 represents the interrupting electrode of a make that is very objectionable, in my opinion. In this the current is broken by pressing down upon the handle, and then, when the finger is taken off, the circuit is made, so that the current is turned on unless there is pressure made upon the trigger; but in delicate manipulations it may be very tiresome to keep the finger pressed upon the trigger when it is not desired to make a circuit.

About these electrodes certain matters of practical detail should be thoroughly understood. The binding-screws should be first carefully looked to. A split holder, into which the end of the wire is to go, is very untrustworthy, because, as has been said already, its elasticity diminishes very quickly, and it does not hold the wire reliably. A hole, through which the wire can be pushed so that a screw can be screwed down upon it and hold it in place, is always the best method, and the screw should be a strong, well-made one. In those electrodes which have handles, the handles should be of a generous length, and should not be less than eight inches in length. They should be sufficiently thick to be grasped by the hand and held without undue effort in flexing the fingers, and they should be hollowed out somewhat on the shank for this purpose and allowed to bulge well at the upper end. The handles of almost all the electrodes that are sold with the batteries are radically defective in these two details of the binding-screw and the length and thickness of the handle.

METHOD OF APPLYING THE BATTERIES.

Let us suppose that, having provided a galvanic and faradic battery, it is desired to know how to make application to the human body, and for this purpose let us take the galvanic battery and speak of some of the ordinary applications. Our apparatus is arranged as in Fig. 81, so that the rheostat and the milliampèremeter are in the circuit. We now attach the electrodes that are needed to the

wires coming off from the binding-posts of the current-collector. Let us suppose that we desire to pass a galvanic current through the brain of a human being. We then attach to one of these wires the electrode of Fig. 67, and this is to be placed upon the neck. We then attach to the other wire the electrode of Fig. 68 or 69; this is to be placed upon the forehead. The electrodes will have been covered with absorbent cotton in the way that has been described, so as carefully to prevent any contact of the skin with the sharp edges of the copper. The skin of the neck and forehead is now carefully wetted with the sponge, wrung out of hot water so as not to drip. The electrodes are also thoroughly moistened with hot water, and quickly squeezed out, so that they will not become dry or cold. The neck-electrode is then put in place, and curved carefully in such a way as to follow the muscular conformation of the neck without unpleasant pressure, and yet so that it shall set firmly. The physician then places the other electrode

FIG. 81.

Battery table showing arrangement of galvanic and faradic apparatus, rheostat, and milliampèremeter.

carefully over the brow, gently shaping it. These two electrodes are now committed to the care of some person who has a steady hand, and who is carefully instructed to place the hand upon the neck-electrode firmly and yet gently, and to hold the forehead-electrode gently and firmly in place by means of the handle. This attendant must not touch the copper of the two electrodes, or else the circuit will be diverted through his or her body, and a shock will be given to the patient. When the electrodes are in place, the current-collectors are turned on to the binding-posts

so as to permit the passage of a current. Now the screw of the rheostat is gently screwed down until it is felt that some resistance is being encountered to its revolutions, and then the screwing should be done very carefully, whilst the eye is kept upon the milliampèremeter to see by the fluctuation of the needle whether the current has begun to pass. It should be remembered that very slight pressure upon the milliampèremeter may cause a difference of one or two milliampères, and if this is made suddenly the patient will perceive a flash of light or experience a slight vertigo. A current of one or two milliampères is now turned on; never more than this at first, although in the course of time it may be increased to three or four. The current is then allowed to pass quietly for three minutes at first, and afterward, as the patient becomes accustomed to it, for a greater length of time. During the passage of the current the person holding the electrodes should not allow the hand to move at all, because a very slight movement will cause an unpleasant shock or a sensation of vertigo. In this cerebral galvanization patients often have a copperish taste in the mouth, but this is a matter of no consequence. When the current has been running for three minutes, the screw of the rheostat should be very delicately and very gently unscrewed, allowing the current to diminish, as indicated by the milliampèremeter, by a fraction of a milliampère at a time, until the current has been entirely turned off, and then, and not till then, the electrodes should be taken away. This description should be read and re-read until the details have become familiar, because it is a lack of attention to these little matters that makes the difference between success and failure in cerebral galvanization, and I have heard many men speak of their failures in this respect when courtesy has prevented me from saying that they did not know how to apply the electricity. When the spinal cord is to be taken into the circuit, the electrodes of Figs. 67 to 70 should be used, but it will not be necessary to exercise as much precaution in turning the current on and off as should be observed when the brain is taken into the circuit. When it is desired to concentrate the galvanic current upon a peripheral nerve, the right ulnar we will say, one electrode of Figs. 67 to 70 should be placed upon the right shoulder, and a smaller electrode, Fig. 73 or 74, should be put over the ulnar nerve, as it is indicated in the charts at the end of this chapter. The current should then be turned on as before, although it will not be necessary to be so extremely cautious about it as when the brain is in the circuit, unless the nerve is very tender. In applying the faradic current the procedure is very simple. All that is necessary is to attach the necessary electrodes to the wires and turn on as much current as may be desired.

THE DIAGNOSTIC USES OF ELECTRICITY.

The diagnostic use of electricity depends upon the altered reaction of nerve and muscle to the galvanic and faradic currents. In order to

understand this it is necessary to comprehend the normal reaction of these structures to these currents. If a muscle is to be set into contraction in the human body, it must be done through the nerve leading to it, because it is practically impossible to limit electricity to a muscle without at the same time implicating the nervous filaments in the muscle, and the practical uses of the current in the human body are therefore quite different from experiments upon frogs wherein muscles are bared with merciless impunity. The reaction of nerve and muscle, for this reason, simplifies itself into a reaction of the muscle to stimulation through the nerve. If a nerve of the human body is tested with the two currents, the faradic and the galvanic, in order to produce a muscular contraction by application of the current to the nerve, it will be found that the response of the muscle varies very greatly according to the current used. If the ulnar nerve, for example, is tested by the faradic current, the resultant muscular contraction will be in proportion to the strength of the current, and this muscular contraction will be be practically about the same in strength, whether the negative pole is placed upon the nerve or whether the positive pole is so placed; in other words, excitation of a nerve by the faradic current causes muscular contraction in proportion to the strength of the current, and this muscular contraction is practically the sme with the negative and the positive pole. With the galvanic current, however, the reaction of the nerve and muscle is more complicated. If the current is turned on from the galvanic battery in the manner that has been indicated, one electrode of Figs. 67 to 70 being placed upon the shoulder or upon the sternum, and the interrupting electrode, Fig. 79, being put upon the ulnar nerve, it will be seen that there is a very great difference accordingly as this electrode is made the negative or the positive pole. It will be found that the first contraction of the muscle will come from the application of the negative pole to the nerve; in other words, a current being gradually turned on, and the nerve being first tested with the positive and then with the negative pole, it will be found that the first contraction is produced with the use of the negative pole to the nerve. Nor is this all. Curiously enough, it will be discovered that this contraction with the negative pole will not come whilst the current is passing through the nerve, nor will it come when the circuit is broken, but only when the circuit is made, or closed. Let us understand this more clearly, for my experience in innumerable lectures has taught me that this is a matter about which men are apt to become confused. The one electrode, as we have said, is placed upon the sternum; another electrode, the interrupting one, is placed upon the ulnar nerve. The latter electrode can be made the positive pole or the negative pole by changing the pole-changer of the battery. Besides, this interrupting electrode can make the current by having the trigger-handle pressed down to come in contact with the little button, or it can break the current by withdrawing the finger from the trigger and letting the latter press back. The current now being gradually turned on, and the interrupting electrode being made first the positive and then the negative pole, it will be found that

a muscular contraction comes with the use of the negative pole for some time before it will come with the positive pole, that this muscular contraction with the use of the negative pole is only at the time that the trigger of the interrupting-handle is pressed down and the current is made, and that there is no muscular contraction whilst the trigger is kept in place so that the current passes, or when the trigger is allowed to fly back and the current is broken. In other words, the first muscular contraction results with the negative pole at the closing of the current. This should be read and re-read until it is thoroughly understood, and then only should the reader go on with what I am now about to say. The current is then gradually increased, when it will be found that the muscular contraction becomes more marked with the negative pole at the closing; and after a while, as the current is still gradually increased, it will be found that a muscular contraction can also be produced with the positive pole. This contraction with the positive pole is generally first with the opening of the current, and then, with a little stronger current, at the closing of the current. It will thus be seen that with the galvanic current, as the current is gradually made stronger, the first muscular contraction is with the negative pole at the closing, and then, when a little more is turned on, with the positive pole, generally first at the closing, and then at the opening. If we now go on increasing the current still more, it will be found that the muscular contraction obtained in these three different ways becomes more pronounced. If, however, we keep on increasing the current until it becomes so strong that it is uncomfortable to the patient, we may become impressed with the fact that we have as yet obtained no muscular contraction with the negative pole at the opening of the current; and we can only obtain a contraction with this negative opening when the current has become unpleasantly strong, sometimes not until it is almost unbearably strong, even in healthy persons. This, therefore, is the reaction of the healthy nerve to the galvanic current. First we have the contraction produced only by the negative pole at the closing of the current; second, as the current is made stronger, there is superadded a muscular contraction upon the application of the positive pole, and at the opening and the closing;[1] and third, we only obtain the muscular contraction with the negative pole at the opening when the current has become unpleasantly or unbearably strong. There is therefore a perceptible interval between the strength of the current needed to produce the first contraction with the negative closing, and that required for the contraction with the positive opening and closing, and there is a very marked increase in strength of current needed to produce a contraction with the negative opening. Suppose now that we endeavor to simplify this reaction of the nerve and muscle by a formula. As I have already said, the positive pole is known as the anode, which is usually indicated by the letter A., and the

[1] It is a matter of no importance whether this muscular contraction with the positive pole comes first with the opening or first with the closing, as this varies in healthy individuals.

negative pole is known as the cathode, designated by the letter C. The letter O. will stand for opening, and the letter C. will stand for closing. We can thus represent the foregoing galvanic reaction of nerve and muscle by this formula:

Negative closing = C. C.
Positive opening = A. O.
Positive closing = A. C.
Negative opening = C. O.

In some text-books the final C. is added to all these to designate closing, but this is unnecessary and apt to confuse.

This formula I call the *progression of the poles.* It is the answer in galvanic language of the healthy muscle when asked a question by the healthy nerve. It is very obvious that any interruption in this formula would denote disease, so that if we got C. O. with the same strength of current that gives us C. C., this would be so contrary to what should be observed in the healthy nerve and muscle that it would be conclusive evidence of some pathological process in one or both of them. And, for the same reason, if the ulnar nerve on the right side responded to a gentle faradic current, and on the left side did not respond to the strongest faradic current, it would mean that there was some lesion in the nerve and muscle in the left side. If we examine a nerve that has gradually become impaired, as after section of the sciatic of the frog, or in a traumatic neuritis in the human being, or in the degeneration of certain peripheral trunks resulting from a myelitis of the anterior horns in a child, we will find that there are marked and characteristic alterations in the response of both the nerve and muscle to the galvanic and faradic current. For these alterations Erb has suggested the significant name of reaction of degeneration (Entartungsreaction). Let us study the peroneal nerve after a sudden onset of paralysis in a little child, in whom a diagnosis has been made of myelitis of the anterior horns. In the first week or ten days there will be diminishing response to galvanism and faradism, so that it will take increasingly larger currents to cause a muscular contraction through galvanization and faradization of the affected peroneal nerve than it does in its fellow of the unimpaired side. In the first few days, when the galvanic and faradic currents are applied to the nerve itself, the decreasing muscular contraction day by day shows that the nerve-excitability to galvanism and faradism is diminishing, and this may go on to total loss of response to both currents in a severe case toward the end of the second week. If the current during this time is applied directly to the muscle, it will be found that the muscular response to faradism is rapidly diminishing and may be entirely lost toward the end or beginning of the second week, as has been said; but the muscular response to galvanism is not nearly so much diminished as is the response to faradism, and, indeed, toward the end of the second week the muscular response to galvanism begins actually to *increase.* This increase of muscular response to galvanism, which is known as

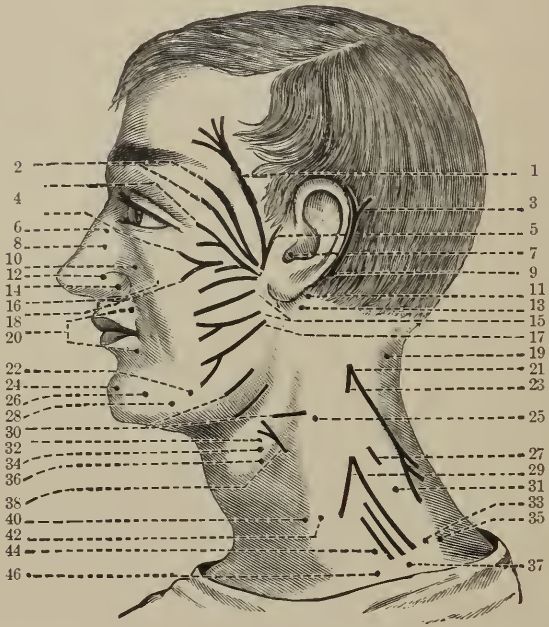
the quantitative reaction of degeneration, rapidly augments, so that at the end of the second week it may be extremely marked. Simultaneously with this increase of muscular response to galvanism goes an alteration of the normal polar reaction, or what I have called the progression of the poles, so that A. C. may equal C. C., and after a while C. O. may equal A. O., or C. O. may even come with a less current than A. O. Simultaneously with this quantitative reaction of degeneration and this alteration of the polar progression there is a change, in response to galvanism, of the character of the muscular contractions, which are quick and shock-like in the normal muscle, but which, at the stage we are describing, become sluggish and worm-like. It is therefore evident that the reaction of degeneration is characterized by a diminution or loss of the faradic and galvanic excitability of the nerve and the faradic excitability of the muscle; whilst the galvanic excitability of the muscle remains—indeed, is often increased—the normal progression of the poles is altered, and the muscular contraction is sluggish and worm-like. In some cases all these different phenomena of the reaction of degeneration may be obtained, whilst in others some of them may be lacking.

In the cases in which there has been a permanent injury inflicted upon the nerve and the muscle, this reaction of degeneration becomes permanent and will be found year after year. In cases which recover, however, the different degenerative phenomena may gradually disappear until the nerve reacts in a normal or almost normal way to the two poles. As a matter of prognosis, therefore, the extent and duration of the reaction of degeneration become of great importance. If a nerve has been injured, and shows only a slight loss of response of nerve and muscle to the galvanic current at the end of two or three weeks, and there is no increased response to galvanism, no alteration in the progression of the poles, and but slight alteration in the character of the muscular movements in response to the currents, the prognosis will be a very favorable one; whereas the opposite of these phenomena would be of very unfavorable augury. Between these two extremes there are all grades. Even in some cases, as I have repeatedly seen, there may be an excellent return of motor power in a nerve and in a muscle which do not yet respond in a perfectly normal way to galvanism and faradism.

In all cases where reaction of degeneration is found, either complete or partial, it indicates disease in the nerve and in the muscle such as is described in neuritis, although the neuro-muscular alteration may be of central origin. It must be remembered, however, that there are certain diseases of the muscles, as in pseudo-hypertrophic muscular paralysis, in which there is scarcely any alteration to galvanism and faradism.

CHART OF THE MOTOR POINTS OF THE DIFFERENT REGIONS OF THE BODY.

FIG. 82.



A diagram of the motor points of the face, showing the position of the electrodes during electrization of special muscles and nerves. The anode is supposed to be placed in the mastoid fossa, and the cathode in the part indicated upon the diagram. (From VON ZIEMSSEN.)

1. Occipito-frontalis (ant. belly). 2. Corrugator supercilii. 3. Occipito-frontalis (post. belly). 4. Orbicularis palpebrarum. 5. Retrahens et attollens aurem. 6. Pyramidalis nasi. 7. Facial nerve. 8. Lev. lab. sup. et alæ nasi. 9. Deep posterior auricular branch of facial nerve. 10. Lev. lab. sup. propr. 11. Stylo-hyoid. 12. Dilator naris ant. 13. Digastric. 14. Dilator naris post. 15. Buccal branches of facial nerve. 16. Zygomat. minor. 17. Subcutaneous branch of inferior maxillary nerve. 18. Zygomat. major. 19. Splenius capitis. 20. Orbicularis oris. 21. External branch of spinal accessory nerve. 22. Branch of levator menti and dep. ang. oris. 23. Sterno-mastoid. 24. Levator menti. 25. Sterno-mastoid. 26. Dep. lab. infer. 27. Levator anguli scapulæ. 28. Dep. ang. oris 29. Phrenic nerve. 30. Subcutaneous nerves of neck. 31. Posterior thoracic nerve to rhomboid muscles. 32. Sterno-hyoid. 33. Circumflex nerve. 34. Omo-hyoid. 35. Posterior thoracic nerve to serratus magnus. 36. Sterno-thyroid. 37. Branch of brachial plexus. 38. Branch for platysma. 40. Sterno-hyoid. 42. Omo-hyoid. 44, 46. Nerves to pectoral muscles.

Fig. 83.

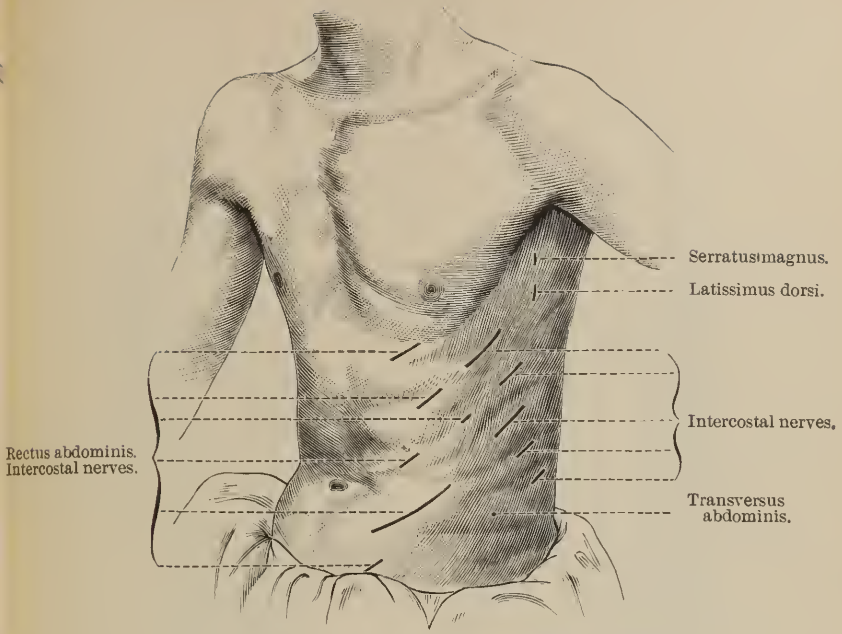


Diagram of the motor points of the trunk. (From Von Ziemssen.)

Fig. 84.

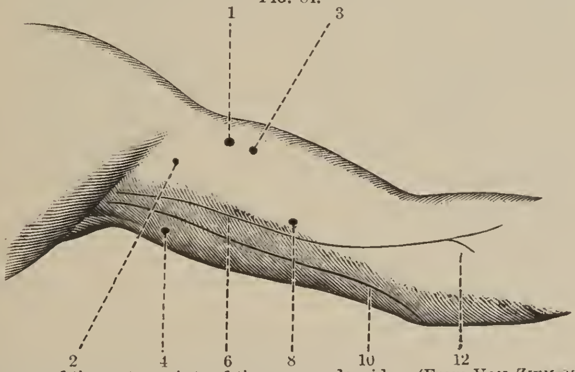
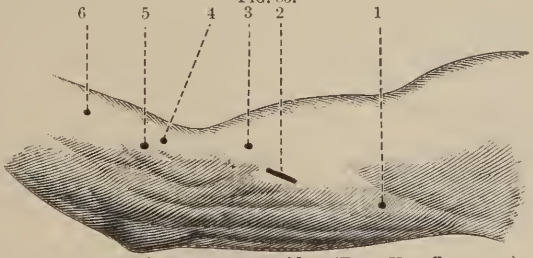


Diagram of the motor points of the arm, under side. (From Von Ziemssen.)

1. Musculo-cutaneous nerve. 2. Musculo-cutaneous nerve. 3. Biceps. 4. Internal nerve of triceps. 6. Median nerve, 8. Brachialis anticus. 10. Ulnar nerve. 12. Branch of median nerve to the pronator teres.

Fig. 85.



Motor points of the arm, outer side. (From Von Ziemssen.)

1. External head of triceps. 2. Musculo-spiral nerve. 3. Brachialis anticus. 4. Supinator longus. 5. Extensor carpi radialis longior. 6. Extensor carpi radialis brevior.

Fig. 86.

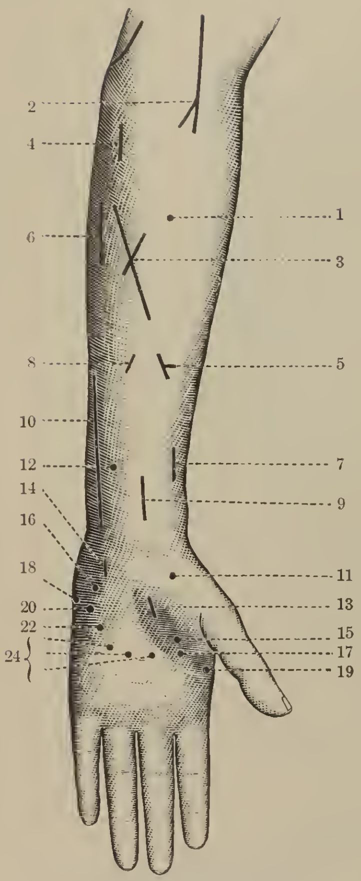
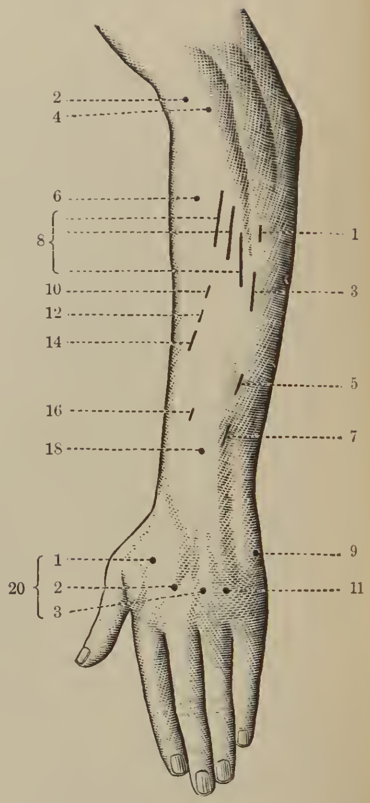


Fig. 87.



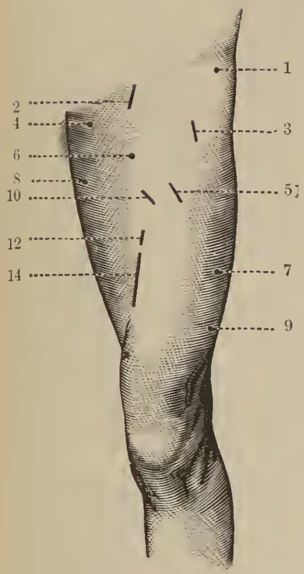
Motor points of forearm, inner surface. Motor points of forearm, outer surface.

(From Von Ziemssen.)

Fig. 86.—1. Flexor carpi radialis. 2. Branch of the median nerve for the pronator teres. 3. Flexor profundus digitorum. 4. Palmaris longus. 5. Flexor sublimis digitorum. 6. Flexor carpi ulnaris. 7. Flexor longus pollicis. 8. Flexor sublimis digitorum (middle and ring fingers). 9. Median nerve. 10. Ulnar nerve. 11. Abductor pollicis. 12. Flexor sublimis digitorum (index and little finger). 13. Opponens pollicis. 14. Deep branch of ulnar nerve. 15. Flexor brevis pollicis. 16. Palmaris brevis. 17. Adductor pollicis. 18. Adductor minimi digiti. 19. Lumbricalis (first). 20. Flexor brevis minimi digiti. 22. Opponens minimi digiti. 24. Lumbricales (second, third, and fourth).

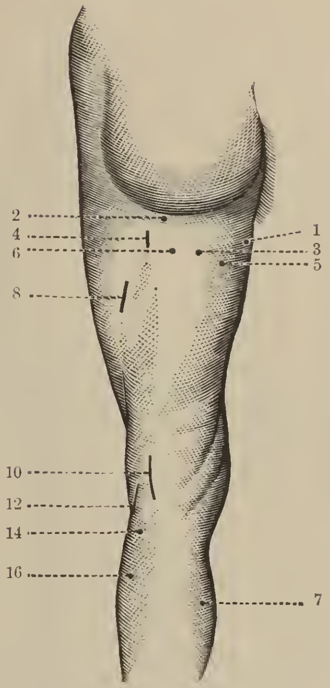
Fig. 87.—Extensor carpi ulnaris. 2. Supinator longus. 3. Extensor minimi digiti. 4. Extensor carpi radialis longior. 5. Extensor indicis. 6. Extensor carpi radialis brevior. 7. Extensor secundi internodii pollicis. 8. Extensor communis digitorum. 9. Abductor minimi digiti. 10. Extensor indicis. 11. Dorsal interosseus (fourth). 12. Extensor indicis and extensor ossis metacarpi pollicis. 14. Extensor ossis metacarpi pollicis. 16. Extensor primi internodii pollicis. 18. Flexor longus pollicis. 20. Dorsal interossei.

FIG. 88.



Motor points of thigh, anterior surface.

FIG. 89.



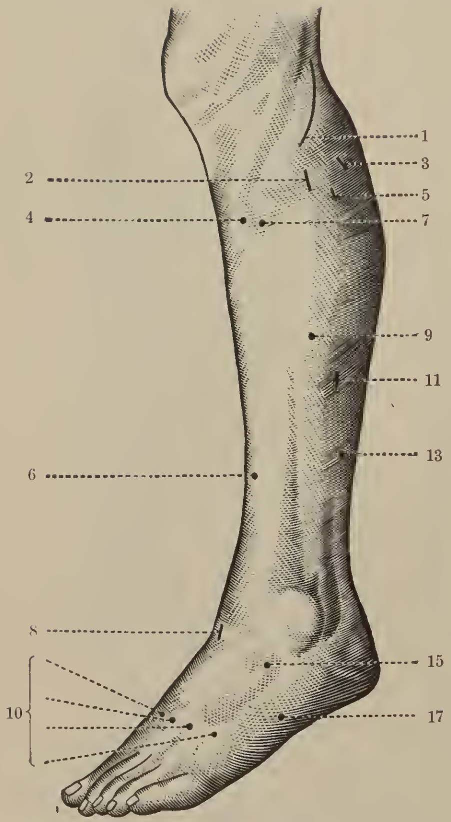
Posterior surface.

(From VON ZIEMSSEN.)

FIG. 88.—1. Tensor vaginæ femoris (branch of superior gluteal nerve). 2. Anterior crural nerve. 3. Tensor vaginæ femoris (branch of crural nerve). 4. Obturator nerve. 5. Rectus femoris. 6. Sartorius. 7. Vastus externus. 8. Adductor longus. 9. Vastus externus. 10. Branch of crural nerve to quadriceps extensor cruris. 12. Crureus. 14. Branch of crural nerve to vastus externus.

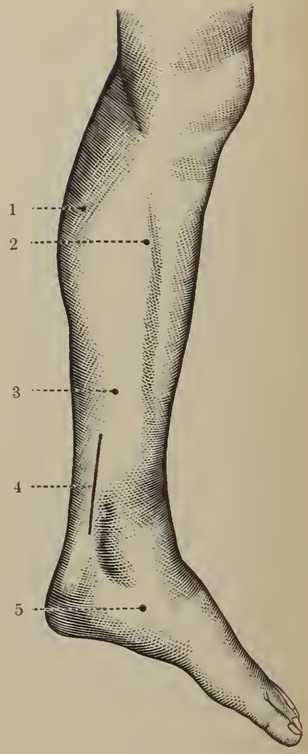
FIG. 89.—1. Adductor magnus. 2. Inferior gluteal nerve for gluteus maximus. 3. Semitendinosus. 4. Great sciatic nerve. 5. Semi-membranosus. 6. Long head of biceps. 7. Gastrocnemius (internal head). 8. Short head of biceps. 10. Posterior tibial nerve. 12. Peroneal nerve. 14. Gastrocnemius (external head). 16. Soleus.

FIG. 90.



Motor points of the leg, outer side.

FIG. 91.



Inner side.

(From VON ZIEMSSEN.)

FIG. 90.—1. Peroneal nerve. 2. Peroneus longus. 3. Gastrocnemius (external head). 4. Tibialis anticus. 5. Soleus. 6. Extensor longus pollicis. 7. Extensor communis digitorum longus. 8. Branch of peroneal nerve for extensor brevis digitorum. 9. Peroneal brevis. 10. Dorsal interossei. 11. Soleus. 13. Flexor longus pollicis. 15. Extensor brevis digitorum. 17. Abductor minimi digiti.

FIG. 91.—1. Gastrocnemius (internal head). 2. Soleus. 3. Flexor communis digitorum longus. 4. Posterior tibial nerve. 5. Abductor pollicis.

CHAPTER III.

MASSAGE.

The manipulations employed in massage are four in number, namely:

1. Stroking;
2. Rubbing;
3. Squeezing;
4. Striking.

The stroking should be in the direction of the venous current—*i. e.*, toward the heart. It is done by varying degrees of pressure with the flat of the hand, its ulnar or radial edge, with the ball of the thumb, with the base of the hand, and with the thumb and forefinger, either one hand being used, or both, as when one completely encircles an extremity.

Rubbing is with the dorsal side of the thumb or with the last phalanx of the middle three fingers. The finger-tips should move in small circles over a small area, but the direction is not important.

Squeezing is performed by grasping the tissues between the thumb on one side and the other fingers on the other, bringing the fingers of the two hands closely together, and making a series of pinchings or kneadings. The tissues may be lifted somewhat out of their ordinary position; or the one hand should be placed on the outer side of a limb and the other on the inner, and all the tissues raised together and wound around the bones, as it were. This squeezing calls for a good deal of muscular strength and practice.

Striking is by blows given by the flat of the hand at its ulnar edge, by the tips of one or more fingers, by the dorsal surface of the fingers spread apart, or by the side of the fist.

This simple division of the manipulations employed in massage is quite sufficient for all practical purposes. The technique, however, has been refined to a marvellous degree, and some of the refinements are no doubt useful in massage of certain delicate organs, but what has been described above is quite sufficient for all general purposes.

Of these different methods, stroking, rubbing, and squeezing are the ones which are most useful, and should be generally employed in cases of nervous and mental disease. Striking is seldom necessary. When the massage is general it should be begun at one foot. This should be grasped with both hands and treated with squeezing and rubbing, at the same time that the thumbs are pressed into the depressions between the bones. The leg should then be stroked and rubbed with a long, rubbing motion, and the different groups of muscles should be squeezed or kneaded, and then the leg should be

given a final stroking all over. The thigh should be treated in a similar manner, and then the lower extremity. The massage should next be applied to the back in the same manner, and the chest after that. In treating the abdomen the procedure should be as follows: The patient should raise his head slightly, bend his legs, breathe freely, and relax the abdominal muscles—which latter, by the way, will often need some little practice. The masseur should then place the tips of the middle three fingers, held closely together, upon the abdominal wall, and rub in small circular movements. The stomach can be reached by making these manipulations in the gastric and left hypochondriac regions; the large intestine by beginning at the cæcum, following the ascending and descending portions of the colon and the sigmoid flexure down to the symphysis pubis; and the small intestine by similar movements in the umbilical and lumbar regions. The upper extremities should then be gone over in the same manner as that in which the lower has been treated.

Simple as these manipulations seem, it yet requires considerable practice to do them properly. In all cases the limb should be put in such a position as to relax the muscles, which can be done by anybody endowed with ordinary intelligence. Certain physical qualifications are necessary in the person giving massage. He or she must, for example, have a certain amount of physical strength so that they can make the squeezings with sufficient force, in order, on the one hand, to manipulate the squeezed tissue properly, and, on the other, to do it with a steady, strong muscular movement that will not be painful. The movements of the masseur or masseuse should under no circumstances be painful. Then, too, a dry and firm hand is an absolute requisite for a masseur or a masseuse. Some authors advise the use of some oleaginous substance wherewith to anoint the skin, but this is never necessary unless it is desired to make use of this unguent for purposes of nutrition. Firm hands, steady movements, and practice make a fat of any kind upon the skin totally unnecessary, and it is only uncleanly. There is no doubt whatever but what the general circulation and the temperature of tissues can be greatly increased by massage, and also that the strength of the muscles can be increased to a degree only short of that of actual volitional exercise. In cases of individuals who for various causes are unable to use their muscles, or in others who are necessarily confined to their room or to bed, massage is an invaluable therapeutic agent. The great difficulty about it is that there are very few persons expert in the giving of it. For several years I almost abandoned its use for this reason, and it has only been since I have kept in my employ a masseur and a masseuse that I have been able to rely upon it. In some few instances I have known of physicians instructing some nurse so as to obtain very creditable results.

In no instance should massage be begun at bedtime, because there are many people for whom the first manipulation means an absolutely sleepless night. In most cases, however, they soon become accustomed to it at any hour. The first manipulation should never extend

beyond a half-hour, and then as the patient becomes accustomed to it, and he has a beneficial result, the time should be lengthened to forty-five minutes, and finally to an hour. The patient should always lie in bed for an hour after the massage, and even longer where there is much weakness.

The directions for the use of massage in individual diseases will be given in the different chapters.

PART II.

NERVOUS DISEASES.

CHAPTER I.

LOCALIZATION OF LESIONS IN THE CEREBRUM, CEREBELLUM, AND SPINAL CORD.

THE diagnoses of the different lesions of the brain and spinal cord are treated of in their proper chapters in this book, but I propose to outline briefly the localization of them in this chapter, so as to obviate the necessity for repetition.

LOCALIZATION OF CORTICAL LESIONS.

As we have seen, the centres which have so far been localized in the cortex are the motor areas, the centres for vision, word-deafness, word-blindness, motor aphasia, and aphasia of interrupted conduction, and possibly also the centres for agraphia and the muscular sense, pain, and touch.

THE MOTOR AREA.

The lesions of the motor area usually produce monoplegic paralysis; *i. e.*, they affect one limb or a group of muscles. It is very important, from a diagnostic point of view, that this fact should be remembered. The reason for it is very apparent. The centre for the leg is contained, as has been said, in the upper fourth of the motor area, that for the arm in the middle two fourths, and that for the muscles of the head and face in the lower fourth, whilst the centre of the motor mechanism of speech is to be found in the third frontal convolution. These different centres are therefore spread over a considerable area of the cortex, and in order that they should all be implicated by one cortical lesion, this would have to extend over the ascending parietal and frontal convolutions and over the third frontal. As the nerve-fibres, however, pass from these various centres toward the base of the brain, they must be converged into the narrowed strait of the internal capsule, as is seen in Fig. 81, and, packed together in this narrow inlet, they can be caught by a very small lesion, such as may be due to a pin-head hemorrhage from the bursting of a capillary bloodvessel. Besides producing monoplegic

paralysis, the lesions of the motor area have a tendency to cause convulsion, which is either localized throughout the entire limb that has been or is to be paralyzed, or else the convulsion starts in this, and, after being confined to it for a distinct though short period, extends to the other members of the same side, or may even become generalized. It has been asserted that when a convulsion due to cortical disease begins in one limb, this is always evidence that the lesion is in the cortical centre of this limb, although the convulsion may extend to the other extremity or to other muscles. This so-called primary convulsion has been called by Seguin the signal-symptom. But it is not always proof that the centre of the convulsed limb is alone affected, for in some cases in which several centres are affected by a lesion the convulsion will commence in one of them and then extend to the others. Nevertheless, when the convulsion has been for weeks or months confined to one limb, and is evidently due to cortical disease, it is almost invariably the case that the centre of the limb primarily convulsed was the first one attacked by the lesion. It was for some time supposed that loss of consciousness, with a more or less localized convulsion, preceded or followed by monoplegic paralysis, was diagnostic of a cortical lesion as distinct from a subcortical lesion; but this has been proven by further cases of my own not to be a reliable criterion, as some cases of cortical lesion may be attended by no loss of consciousness, whilst some cases of subcortical lesion are. The diagnosis, therefore, of a cortical lesion of the motor area is to be made mainly by means of the presence of a monoplegic paralysis, with or without monoplegic convulsion, and exclusion of subcortical areas.

CORTICAL CENTRE OF VISION.

As we have already seen (Fig. 39 and Plate I.), the cortical centre of vision is in the cuneus and the adjacent occipital convolutions; and the defect of sight produced by the lesion in this centre is that which is known as hemianopsia. We shall have occasion to examine subcortical lesions also capable of producing hemianopsia.

CORTICAL CENTRE OF WORD-BLINDNESS.

This, as we have seen (Plate I.), is supposed to be located in the angular gyrus, although there is still some doubt about the matter.

CENTRE OF WORD-DEAFNESS.

Recent cases by Mills have demonstrated that this peculiar lesion is located more particularly in the rear ends of the first and second temporal convolutions, so that we can readily understand why it is that word-deafness is so often associated with word-blindness.

CENTRE OF AGRAPHIA.

As has already been stated, agraphia is an inability to write, although there may be no paralysis of the hand, no loss of the memory

of words, no word-blindness, and no hemianopsia. Its centre is not positively known, but is supposed to be in the posterior portion of the second frontal convolution.

CORTICAL CENTRE OF APHASIA.

When the third frontal convolution is affected, the patient suffers from what used to be known as ataxic aphasia, but what has recently been termed motor aphasia—*i. e.*, the patient understands perfectlt well the meaning of words and can recollect them well enough, buy he cannot pronounce them properly. When the island of Reil is affected there is what is known as aphasia of conduction—*i. e.*, the patient utters words entirely unlike those which he had intended to speak, and, being thus unable to express himself with the words which he had desired to use, seemingly gives vent to gibberish.

A sharp distinction must be made between four varieties of defect of expression—namely, motor or ataxic aphasia, aphasia of conduction, word-deafness, and agraphia. The patient who has motor or ataxic aphasia can remember words perfectly well, as is evident by his being able to write them, being able to express his appreciation of their meaning in writing, and by being able to make some defective attempt at the pronunciation of them. The patient who has aphasia of conduction can also appreciate the meaning of words perfectly well, as is shown by his being told to do a certain thing and his performance of the act in a perfectly intelligent manner; and he can also write the words which are given to him, but he cannot make even a defective attempt at the pronunciation of them, but simply utters some word that has no relevancy. The patient who has word-deafness can pronounce a word perfectly well, and repeat it when it is pronounced by another, so that he has evidently no defect in the mechanism of speech; and he can also write the word which he is told to write, showing that there is no fault in the mechanism necessary to writing; but he does not understand the meaning of the word which he repeats. You can say to him, for example, "This is your watch," and he will repeat the word "watch," and will also write it; but if asked the meaning of it, not only will his facial expression evidence that he does not know what it means, but he will confess, if he is intelligent, that he does not know the meaning of "watch." If, however, he is shown the watch, and often when he has written the word "watch," his face will brighten up with a look of intelligence, and he will tell you that he knows what you mean; in other words, through the sense of sight he has gained a comprehension of the meaning of the word by seeing the article. He can also gain an idea of the meaning of the word by other senses than sight, as by the sense of touch in feeling the watch, or the sense of smell in smelling a flower, or the sense of taste in tasting an object, or by the muscular sense, when he is asked the meaning of the word "forefinger," and you move his forefinger, or by the sense of pain when you ask him the meaning of the word "pin," and then prick him with one. Finally, when a patient has agraphia he is not able to write,

although he may be able to understand the meaning of words perfectly well, may be able to pronounce them properly, may have a perfect memory of them, and may have no word-blindness, hemianopsia, or paralysis of the hand.

THE CENTRES OF MIND ALONE.

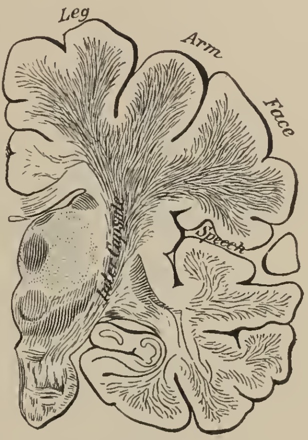
It was formerly held that the frontal lobes anteriorly to the motor area were the only centres of mind—*i. e.*, that only a lesion of them could produce mental symptoms purely, without any such as are due to implication of the centres of the limbs, sight, hearing, and aphasia. Even at the present day is an idea of this kind dimly floating in the neurological mind; but as cases multiply, this doctrine is gradually being disproved, and it is being shown more and more clearly that the metaphysical entity which we call mind is dependent upon the structural integrity of the cortex as a whole, so that a lesion anywhere in the cortex, if sufficiently destructive, may produce mental symptoms, and the more widespread such it is, the greater will be the mental impairment, although a small area of disease may seem to a superficial observer to leave the mind unimpaired, when careful examination of the patient will show that this is only seemingly so. Nevertheless, it remains true that involvement of the frontal lobe anterior to the motor area and the third frontal convolution produces only mental symptoms, and not physical ones. Some cases, notably the celebrated crowbar one, have been vaunted in contradiction of this; but an analysis of the history has shown that there were mental changes sufficient to prevent the individual from following his occupation, whilst subsequent cases have abundantly proved the statements just made.

LESIONS OF THE CENTRUM OVALE.

The centrum ovale, it should be remembered, is nothing more than the nerve-strands coming down from the cortex to pass to the sublying ganglia, and lesions of it therefore do not differ from lesions of the cortex except in the fact that the deeper the level of the lesion, the more convergence and interlacing will there be of different nerve-fibres coming from different cortical centres. (See Fig. 92.) Thus, a lesion just below the centre for the leg and sufficiently localized to affect only the nerve-strands coming from it, would cause exactly the same symptoms as a lesion of the leg-centre itself—viz., paralysis or convulsion, or both, of the leg. If the lesion were still deeper seated, so as to implicate the nerve-strands coming from the leg and the arm-centres and converging so as to reach the internal capsule, the symptoms produced would be that of simultaneous affection of the arm- and the leg-centres, namely, paralysis or convulsion, or both, of the arm and leg. The deeper down we go into the centrum ovale, therefore, the more complex will be the symptoms. It is often extremely difficult to make a diagnosis between a cortical and a subcortical lesion, and we

are generally obliged to seek aid from clinical facts as well as anatomical ones. Thus, if there is a monoplegic paralysis and convulsion at the start, and this paralysis and convulsion remain monoplegic through months, there is a greater probability of the lesion being cortical than of its being subcortical, for the reason that there would be more space in the cortex for the lesion to develop and affect only one limb than there would be in the centrum ovale. Again, if the diagnosis of a tumor can be made, and the symptom is a paralysis or convulsion which has remained monoplegic for months, the probability is greatest of the lesion in the cortex, because a tumor in the centrum ovale would be likely to implicate nerve-fibres from other centres more quickly in its growth. Again, if the lesion has been a trauma of the skull, the probability is greatest of the trauma having impinged upon the cortex rather than upon the subcortical regions. Again, if a diagnosis has been made of a meningitis, it is certain that the cortex would be affected by the meningitis long before the subcortical tissue was implicated. Again, if a diagnosis can be made of an arterial lesion, as in a condition of general endarteritis with attendant atheroma or embolism, the probabilities are far greater of the centrum ovale being implicated, because the vessels from the pia mater inosculate freely in the cortex, whilst the subcortical matter is fed with relative sparseness by long, straight arterial branches dipping down into it. (Fig. 93.) When, however, the paralysis is not monoplegic, as when both limbs on the same side are affected, or when there is an affection of several limbs, or of the muscles of the head and trunk with coincident aphasia, it may be a very difficult matter to make the diagnosis beween a cortical and subcortical lesion, and in most cases it is almost impossible. To sum up, therefore:

FIG. 92.



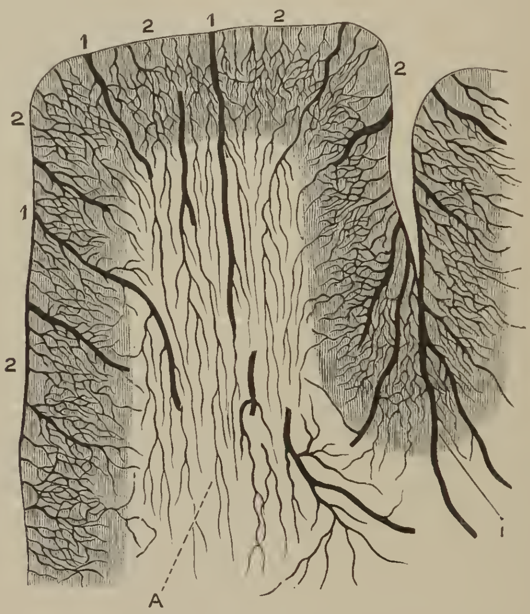
Cortical centres and subcortical nerve-strands leading up to them.

1. A cortical lesion is most probable when one centre alone is affected, and remains affected. Thus, if there should be paralysis of one arm, with or without convulsion limited to that one arm or starting in it, the probability would be exceedingly great that there was a lesion in the middle two fourths of the motor area. So, also, when there are paralysis and convulsion of one leg, or of the muscles of the head and face, or aphasia alone, or word-deafness alone, or agraphia alone, or hemianopsia alone, it would be extremely probable that the lesion was cortical in the respective centre. If, moreover, there should be a paralysis of a few muscles in one limb, as of the thumb, and this paralysis should remain for some time before other

symptoms supervened, the probability would pass into certainty of there being a cortical lesion.

2. A cortical lesion would be rather more probable than a subcortical one when there were persistent attacks of loss of consciousness with the localized symptoms, although, as I have said, the weight to be attached to this loss of consciousness is rather problematical.

FIG. 93.



Distribution of arteries to gray and white matter. (DURET.)

1, 1. Medullary branches passing directly through the gray matter to the white matter, and then terminating in branches that do not anastomose with their neighbors ("terminal arteries"). 1. Medullary arteries in the sulcus between two convolutions. 2, 2. Cortical arteries.

3. A cortical lesion is most probable when one centre is affected first, and then, a distinct interval of time having elapsed, another centre is affected, and still more if there is a progression of symptoms to a third centre, and so on. But this must be nevertheless taken into consideration: If centres are closely approximated, as are those of the arm and leg, the head and neck, and those of motor aphasia and aphasia of conduction, the subcortical nerve-strands from these centres are also close together, so that a growing lesion could readily extend subcortically as well as cortically. But if centres as far apart as those for the leg and hemianopsia should be affected in progression, it would then be most probable that the lesion in both cases was in the cortex, and multiple, because it would be impossible to conceive of a subcortical lesion that should extend from beneath the upper third of the motor area away back to just beneath the occipital lobe without causing enormous general symptoms, not only of implication of the whole cerebrum from such

a large lesion, but also from impairment of various nerve-centres between the two main ones which we have named.

4. Meningitis and traumata are more likely to affect the cortex than the centrum ovale.

5. Tumors are somewhat less likely to affect the centrum ovale than the cortex, although subcortical tumors are found often enough to prevent this fact from being conclusive.

6. Cortical tumors and meningitis, so far as we now know, are alone capable of causing a distinct rise of cerebral temperature, as indicated by my cerebral thermometry. This fact, it should be borne in mind, has only the positive value of making a distinct rise of cerebral temperature probably indicative of a tumor or meningitis, but, conversely, the absence of rise of cerebral temperature is of no value for or against.

This difficulty of diagnosis between a cortical and a subcortical lesion is of very great importance to the surgeon. In any case in which it cannot be made many a sore disappointment will be avoided by carefully explaining to the patient and the friends the uncertainty of the localization of the lesion. To this difficulty should be added the further one of finding the exact site of a lesion in the centrum ovale, even when it has been localized there. This, however, will be dwelt upon fully in the chapter upon intra-cranial growths (Chap. VII.).

LESIONS OF THE INTERNAL CAPSULE.

Lesions of the internal capsule are almost invariably due to arterial trouble, either of the nature of endarteritis, embolism, or thrombosis. In Fig. 55 it has been shown how the terminal arterioles coming off from the Sylvian artery supply the internal capsule. At page 59, the location of the different nerve-strands in the internal capsule has been described. Just behind the knee or genu (Fig. 35) lie the fibres from the motor area, the so-called pyramidal tract. Immediately in front of them run the nerve-strands from the facial and hypoglossus centres, which probably are situated, as we have seen in Plate I., in the lower portion of the ascending frontal convolution. Behind the pyramidal tract in the posterior third of this portion of the internal capsule run the sensory fibres, which are on their way to the tegmentum. Back of the sensory fibres are the optic-nerve fibres coming in from the occipital lobe. Somewhere in this region are also fibres from the temporal lobes to the auditory nuclei, and others from the olfactory lobes. Besides these main motor and sensory fibres there are other short ones whose existence we know of anatomically, the lesions of which produce symptoms that are not known to us. Anteriorly to the knee or genu, there are fibres from the frontal lobe to the pons, and from the cortex to the optic thalamus; whilst in the posterior or sensory portion of the internal capsule, behind the genu, are fibres to the optic thalamus that are probably sensory, and from the temporo-occipital cortex to the pons. Lesions, therefore, of the internal capsule produce either motor or sensory symptoms, or

commingled motor and sensory symptoms. In the majority of cases, however, the symptoms are motor and but slightly sensory, and the portion of the internal capsule affected is that supplied by the lenticulo-striated artery. (Fig. 55.) The form of paralysis is hemiplegic; *i.e.*, paralysis of motion or sensation, or both, most frequently motor alone, of the upper and lower extremity and of the lower part of the face, the eyelids and eye-muscles being unaffected. (See p. 85.) The diagnosis of a lesion of the internal capsule is readily made. As has been said, the paralysis is generally motor only, though sometimes both motor and sensory; the onset is relatively acute, being either sudden or extending over a few hours or a day; and in the vast majority of cases the lesion is one due to some arterial trouble, such as an endarteritis, embolism, or thrombosis. The lesion, therefore, occurs mainly in certain classes of individuals: first, those who have the chronic endarteritis of middle age, associated with general atheroma, hypertrophied heart, and chronic degenerative kidney lesion; second, those who have an intracranial syphilis producing syphilitic endarteritis; third, children who have hemiplegia, either without mental defect or with very slight mental impairment; fourth, in rare cases those who have a premature endarteritis from alcoholism; fifth, those who have the endarteritis that is associated with nephritis. Tumors of the internal capsule hardly ever occur, and traumata impinging only upon this narrow strait of the internal capsule are equally rare, except where there is existing arterial disease that renders the arterioles excessively fragile.

LESIONS OF THE CORPUS STRIATUM AND OPTIC THALAMUS.

These cannot be diagnosed. It is possible, however, that the recent localization by Ott of a heat-centre (see p. 103) in the optic thalamus will be the means of our making a diagnosis, although as yet this has not been done, and this centre has only been localized in the dog and the rabbit.

LESIONS OF THE QUADRIGEMINAL BODIES.

As has been seen in Figs. 36, 38, 40, 45, and 48, the quadrigeminal bodies are in close relation to the optic-nerve fibres, to the tegmentum, to the superior and middle cerebellar peduncles, to the pineal gland, to the pulvinar of the optic thalamus, and also to the nuclei and nerve-fibres of the ocular nerves. It can well be seen, therefore, how much doubt there is as to whether the symptoms are due to lesions alone of the quadrigeminal bodies or to associated lesion of the surrounding structures. Such cases, however, as have so far been published, some twenty-five in number, would seem to show that the symptoms are: first, an unsteady, reeling gait appearing early in the course of the symptoms; second, ophthalmoplegia of the muscles of both eyes, but not quite symmetrically, nor implicating all the muscles in equal degree. Nothnagel believes that the reeling gait is due to the quadrigeminal lesion alone, as several cases have been

reported in which the cerebellar peduncles were not affected. Thus Feilchenfeld, Pontoppidan, and Fischer have reported three cases of tumor of the pineal gland alone, with pressure upon the quadrigeminal bodies, whilst Massot has reported a case of a tumor of the pineal gland in which the quadrigeminal bodies were not compressed and there was no reeling gait. The reeling gait is like that of cerebellar lesion, and consists of a stumbling or titubation similar to that of a drunken man, and it is entirely dissimilar to the ataxia of locomotor ataxia, in which the patient walks erectly, but brings his heels down with a stamp or a tendency to a stamp, and the upper extremities are not affected at all. The ocular muscles which are most prone to be affected are those supplied by the third pair, although, as has been already stated, other ocular muscles may be affected so as to give rise to an ophthalmoplegia. The lesion of the ocular muscles is due to compression or extension of the quadrigeminal lesion to the nuclei and nerve-fibres of the ocular nerves. As has been said, this ophthalmoplegia is not apt to be complete or bilateral. Thus, when the third nerve is affected, it is generally the superior and inferior recti which are paretic or paralyzed, or the lateral movement of the eye may be abolished, or ptosis may be the most marked symptom; and although ophthalmoplegia occasionally occurs, it is seldom as complete as when the nuclei themselves are affected (see Progressive Ophthalmoplegia). Nystagmus alone, or in conjunction with paralysis of ocular muscles, may occur. It has also been claimed that the trochlear or abducens nerve alone can be affected, and it was at one time thought that this was a diagnostic symptom, but it has not been proven. Nothnagel has recently expressed the belief that lesions of the quadrigeminal bodies may leave the vision entirely unimpaired, but in making this statement he does not seem to have taken account of any other impairment of sight than amblyopia or amaurosis, the cases which he cites do not seem to have been definitely examined for hemianopsia, and in other cases he admits that choked disk and optic neuritis were present in such degree as to make it impossible to examine thoroughly the visual acuity; moreover, the recent experiments and cases of von Monokow have proven that the anterior quadrigeminal bodies, together with the lateral geniculate body, are connected with the optic nerve-fibres. Diagnosis of a lesion of the quadrigeminal bodies may therefore be made by means of the reeling gait, associated with ophthalmoplegia or isolated paralysis of the trochlear or abducens nerve, or with nystagmus, and also by the absence of other motor, sensory, or vasomotor symptoms. I think it is probable that the future will show that hemianopsia is associated with these symptoms, especially when the anterior quadrigeminal bodies are implicated; and it must not be forgotten that the posterior quadrigeminal and middle geniculate bodies have been shown by von Monokow and Spitzka to be connected with hearing; deafness has been a sign of lesion in this region in a number of cases analyzed by Weinland. Flechsig, Bechterew, Roller, Monokow, and others have claimed, indeed, that a portion of the lateral lemniscus running to the posterior quadrigeminal bodies contains fibres from

the cochlear nerve; but Weinland found the lateral lemniscus totally destroyed on the side opposite the deafness and the diseased quadrigeminal body.

LESIONS OF THE PONS.

Homén, Meyer, and Spitzka have carefully studied cases of lesion of the pons. Of these three, the most minutely and therefore thoroughly described is Spitzka's. In these three cases the age was forty-eight, forty-eight, and fifty-eight, respectively, and the lesion was an arterio-sclerosis causing either hemorrhages or softening. It must be remembered that, as has been shown in Figs. 36, 37, 38, and 48, the pons is a very complicated structure, containing motor and sensory fibres passing from the cortex down to the spinal cord, as well as nerve-fibres and nerve-nuclei of the different cranial nerves, the cerebellar peduncles, and transverse fibres connecting the two lobes of the cerebellum. The exact lesions, therefore, will vary to some extent according to the level of the pons that is affected, but these three cases of Homén, Meyer, and Spitzka, together with the knowledge gained from other sources, have enabled us to speak with some certainty of the main diagnostic symptoms. In the former there were: 1, paralysis of the tongue, which deviated to the side of the lesion; 2, hemianæsthesia of the opposite side; 3, constant dizziness; 4, ataxia of the limbs of the opposite side; 5, exaggerated knee-jerk of the opposite side; 6, diminution of cutaneous reflex of the opposite side; 7, subjective sense of muscular cramping of the extremities of the opposite side. The hemianæsthesia consisted of impairment of touch, temperature, sense of weight, and muscular sense—the latter to a slight degree. The ataxia was greater on closing the eyes, but was not increased by placing the feet close together. In Meyer's case there was a loss of faradic excitability of nerve and muscle in the face, and of galvanic excitability of the nerve in the same region. Meyer's case had also contracture, muscular atrophy, and trophic disturbances of the arm opposite to the lesion. In Spitzka's case the pupil became somewhat contracted nearly three years after the first symptoms appeared, whilst in Meyer's case the pupil was unaffected; and this is a contradiction of the prevailing view that a lesion of the pons must be necessarily accompanied by a contracted pupil, although cases do occur in which there may be myosis, at the same time that other cases have been observed in which they have been found dilated. The reaction of the pupils to light is an equally unreliable symptom for purposes of diagnosis. The lesion in Homén's, Meyer's, and Spitzka's cases implicated the lemniscus, and in Spitzka's case it was about at the level that is figured in Figs. 42 and 43, about at *Lm* and extending downward.

Lesions of the pons may be diagnosed by the following symptoms: 1, paralysis of sensation or motion, or both, of the side of the body opposite to the lesion; 2, paralysis of the trigeminal, abducens, facial, or hypoglossal nerves, and possibly also of the auditory, all upon the same side of the body as the lesion; 3, vertigo; 4, exaggerated knee-jerk on the side of the body opposite to the lesion;

5, subjective sense of cramping of the extremities of the side of the body opposite to the lesion; 6, absence of mental disturbances; 7, possibly conjugate deviation of the eyeballs and head toward the side of the lesion; 8, difficulty of deglutition and articulation; and, 9, ataxia of the opposite limbs. As we have already seen, the pupillary contraction is an unreliable sign, although it is probably of value in the diagnosis of a sudden hemorrhage into the pons, when it has been frequently observed as a temporary symptom. If disease should be situated high in the pons, however, near where the crus passes into it, there would be a unilateral paralysis—*i. e.*, the cranial nerves and the limbs would be affected upon the same side, opposite to the lesion. This can be readily understood when we remember that the nerve-fibres from the different cranial nerves have here reached the side upon which they are going up to the cortex, just as they have in the internal capsule. (Figs. 36 and 46.)

LESIONS OF THE MEDULLA OBLONGATA.

Lesions of the medulla oblongata assume either the form of progressive ophthalmoplegia or labio-glosso-laryngeal paralysis, which are treated of in separate chapters. Lesions of the individual nerve-nuclei are difficult to diagnosticate, unless symptoms of impairment of a cranial nerve are observed in the course of one of these symptom-groups, or of a progressive muscular atrophy.

LESIONS OF THE CEREBELLUM AND ITS PEDUNCLES.

The diagnosis of cerebellar lesions is a very difficult matter. It was formerly supposed that a swaying, staggering gait was pathognomonic, but since it has been shown to be present in the two carefully observed cases of pons lesion recorded by Spitzka and Meyer, this view is no longer tenable. When, however, to the staggering, swaying gait there is added a marked tendency to staggering persistently in one particular direction, this would be indicative of lesion of the cerebellar peduncles, and therefore the probability of a coincident cerebellar lesion would become stronger. These movements in one particular direction may consist in staggering to one side, or backward, or forward, or in a semi-rotary movement around a circle. A peculiar condition of the eyes was observed in one case of Monat's: they were motionless, and one looked downward and outward, the other upward and inward. In three cases of my own I have also observed a symptom to which I have seen no allusion, namely, a peculiarly tight drawing of the scalp over the skull during the paroxysms of headache and dizziness. Many cases of disease of the cerebellum and its peduncles have been latent so far as regards symptoms. It is, therefore, a matter of considerable doubt as to whether we can positively make a diagnosis between lesion of the cerebellum and its peduncles and that of the pons; although the probability would be strongly in favor of a disease of the cerebellum or its peduncles when there existed a swaying, staggering gait, especially when this was

accompanied with forced movements in certain directions, and when there was no motor or sensory paralysis of the extremities and no implication of cranial nerves.

LESIONS OF THE SPINAL CORD.

The symptoms of lesions at the different levels of the cord can be ascertained by means of the table upon page 82. The localization of symptoms in the various portions of the gray and white matter can only be made to a limited extent. Thus, motor paralysis with muscular atrophy and entire absence of sensory symptoms would indicate that the anterior cornua of the cord are implicated, as in poliomyelitis anterior and some forms of progressive muscular atrophy; and a lesion may be localized in the lateral pyramidal column when there are exaggerated tendon-reflexes, contracture, and hasty micturition, as in secondary descending degeneration of this tract from some higher level in the cord, pons, or cerebrum. But a disease of the posterior cornua cannot be diagnosed, nor of such white strands of the cord as the columns of Goll, Burdach, Spitzka-Lissauer, direct cerebellar and Gowers' columns, and the anterior fundamental column. It has been supposed that the posterior columns of Burdach and Goll contained the tactile and muscular sense nerve-fibres, and the lateral and anterior fundamental and the Gowers' column the temperature and pain nerve-fibres, but many clinical observations have failed to prove this view, as well as the belief that lesions of the direct cerebellar columns are always the cause of ataxia in spinal lesions.

LESIONS OF THE PERIPHERAL NERVES.

Lesions of the peripheral nerves can be diagnosed by the symptoms that will be detailed in the chapter upon Neuritis.

HEMIANOPSIA.

At the outset of this subject it should be clearly understood that there is a technical difference in the meaning of the two words hemianopsia and hemiopia, although their etymological derivation is almost the same (*vide* page 41). Hemiopia is the blindness of bilateral halves of the retina. As is well known, the rays of light coming from the outside cross in the optic lens, and therefore a person who has a left-sided hemiopia will fail to see objects to the right side of the median line, and this external blindness is called hemianopsia—*i. e.*, a left hemiopia will cause a right hemianopsia.

As has been shown in the diagram, Fig. 39, page 63, the course of the optic nerve is a very complicated one. The cuneus of the occipital lobe, the adjacent occipital convolutions, and probably also the gyrus angularis and the lingual lobule (Fig. 16) are the cortical centre of sight, and a lesion of the region thus constituted produces a hemiopia of corresponding halves of the retina, or hemianopsia of

the opposite side. From the cortical centre of sight the optic fibres pass through the centrum ovale to the posterior portion of the internal capsule, as shown in Fig. 15. From the internal capsule they then pass through the posterior portion of the optic thalamus, the pulvinar, into the anterior quadrigeminal body, and thence to the lateral geniculate body (Figs. 38 and 39), receiving fibres from adjacent structures. From the anterior quadrigeminal body and the lateral geniculate body the fibres then pass back and across the optic thalamus to the base of the brain into the optic tract (Fig. 39), and through the optic tract to the optic chiasm and thence to the eye. Most of the fibres, as has already been said, pass from the cortical centre of sight through these intermediate structures (optic thalamus, anterior quadrigeminal body, and lateral geniculate body) to symmetrical halves of the retina upon the same side as the cortex from which they have taken their origin. Besides these, however, certain fibres pass to the outer portion of the retina upon the same side as the cortex, and other fibres pass in front of the chiasm to connect the two retinæ. The chief stations, therefore, at which the optic fibres are apt to be implicated are: the cortical centre of sight, viz., the cuneus, the adjacent occipital convolutions, probably also the lingual lobule or the angular gyrus; the centrum ovale, the internal capsule, the region of the corpora quadrigemina, the optic tract, the chiasm, the optic nerve in front of the chiasm, the retina.

A lesion of the cuneus or the other convolution just named is a cortical lesion, and will have the general symptoms of cortical lesions, besides the localizing ones. The latter can be diagnosed by the fact that the blindness is bilateral, affecting the eye on the same side as the cortical lesion, the dividing-line of the blindness being nearly vertical. This variety is called homonymous hemiopia, or hemianopsia. The dividing-line is generally precisely on the vertical meridian, and straight; but this is not always so, for it may be sinuous or oblique or forming an angle, and it is not always the same in each eye. Hun has also shown that the blindness may be in the upper quadrant of each retina on the same side, although Von Monokow believes that several cases warrant him in affirming that these quadrant defects are generally, if not always, due to implication of the anterior quadrigeminal or lateral geniculate bodies. This cortical lesion causing hemiopia may be the only localizing symptom present, but there sometimes coexists word-blindness, due to coincident lesion of the angular gyrus, and mental blindness, due to lesion of the posterior portion of the first and second temporal convolutions (page 41); but there will usually be no other symptoms of cortical implication.

When hemianopsia is due to a lesion of the centrum ovale, the diagnosis may be very difficult, inasmuch as the same symptoms will be present as when the cortex alone is affected. The reason for this will be evident when we consider that the same symptoms are presented in case of a cortical centre or centres being implicated, as when the nerve-strands running from these cortical centres through the centrum ovale are involved; in other words, it is as difficult to make

a diagnosis of a subcortical lesion in this portion of the brain as it is in others.

A lesion of the posterior portion of the internal capsule can be diagnosed with more ease, because at this point with the hemianopsia there will be general sensory symptoms which are known by the name of "hemianæsthesia," namely, impairment of touch, pain, muscular sense, taste, and sometimes temperature-sense, upon one side of the body. But this hemianæsthesia will be upon the opposite side to the hemiopia and upon the same side as the hemianopsia, and in some of these cases of lesion of the internal capsule the motor fibres lying in the anterior part of this latter structure will also be affected, so that hemiplegia will be present.

When there is a lesion in the region of the corpora quadrigemina, conjointly with the hemianopsia will be general symptoms of implication of this region (page 146), namely, a staggering gait and some implication of the ocular muscles. It is claimed that Wernicke's pupillary reflex is of aid in this diagnosis; *i. e.*, in lesions behind the pulvinar, anterior quadrigemina, and lateral geniculate body the pupils will respond to light thrown upon the blind side of the eye, although they act sluggishly; whilst this reaction of the pupil will be absent when the lesion is at these ganglia or in front of them. This symptom of differential diagnosis has been verified by Wernicke, Seguin, and Brieger. Haab has recently called attention to another pupillary reflex which he designates as the pupillary cortical reflex. This is tested in the following manner: A light is placed about forty-five degrees sidewise from the patient, the examination being made in a dark room and care being taken not to throw too much light upon the pupil. The patient is then made to fix one eye of the examiner and to maintain the fixation steadily. After he has done this a short while he is asked if he can see the laterally placed flame of the light, and at the moment when the patient turns his attention to this flame, whilst he keeps his eye fixed upon the examiner's eye, a contraction of the pupil can be distinctly seen in the normal eye. It must be remembered, however, that this examination must not be prolonged, because it is difficult for a person to keep his attention fixed upon the lateral flame; but this length of time can sometimes be increased by having the patient regard some peculiarity of the examiner's eye, and, whilst he is regarding this intently, suddenly turn his attention to the light, when the examiner must watch the pupil. This examination may also be made by placing the flame above or below or at different places. Haab regards this, with seeming reason, as a cortical reflex of the pupil, and believes that it may be of value in making the diagnosis between a cortical lesion and a lesion of the lower structures through which the optic fibres pass. He does not, however, cite any pathological cases; nor, according to a letter recently received from him, does he know of any clinical material in support of his views.

When the lesion is at the base of the brain (Figs. 18 and 36) the optic symptoms will be usually accompanied by those due to lesion of the structures at the base, such as the third nerve especially, or of the

sense of smell from implication of the olfactory, and, when the lesion extends backward, of the pons and the different cranial nerves which take their origin at the sides of the posterior part of this structure. In such cases the hemianopsia is usually bitemporal, as by a lesion at the middle of the chiasm involving the crossing-fibres from both tracts. In some very rare cases the hemianopsia is binasal, and is due to a lesion on both sides of the chiasm or on the outer side of each optic nerve. If there is a lesion of the optic nerve in front of the chiasm, or on the lower or upper part of the chiasm, or of both optic nerves, there will be a blindness of half of the field in only one eye. These lesions at the base of the brain usually include not only loss of sight, but also of color- and form-sense, whilst the preservation of light-sense, with a loss of either color-sense or form-sense, is frequently found with lesion of the cortex or of the region of the corpora quadrigemina.

CHAPTER II.

TESTS OF THE GENERAL MOTOR AND SENSORY SYMPTOMS OF THE NERVOUS SYSTEM.

THE different senses which are to be tested are: sight, hearing, smell, taste, touch, pain, muscular sense, temperature, weight.

SIGHT.

The eye should be tested as to vision, color-sense, condition of the retina, and pupillary reaction.

In a text-book of this kind it would be impossible, of course, to go into all the methods of examination of vision which would be appropriate in a text-book upon the eye, and I need only call attention very briefly to those matters which are of more especial importance in connection with neurology, such as the condition of the pupil, the external ocular muscles, the eyelids, the determination of gross defects of sight, and the state of the retina.

The pupil should be examined to ascertain its size when in repose, the size relatively of the two pupils, the reaction to light, and the reaction to accommodation.

The following table, and most of the text explanatory of it, are taken from Soelberg-Wells, edited by Bull, and will be of use in enabling the reader to remember more easily the manner in which the different movements of the eye are produced:

Movement	*Is produced by the action of the*
Upward.	Superior rectus and inferior oblique.
Downward.	Inferior rectus and superior oblique.
Inward.	Internal rectus.
Outward.	External rectus.
Upward and inward.	Sup. rectus, inf. rectus, and inf. oblique.
Upward and outward.	Sup. rectus, exter. rectus, and inf. oblique.
Downward and inward.	Inf. rectus, inter. rectus, and sup. oblique.
Downward and outward.	Inf. rectus, exter. rectus, and sup. oblique.

In other words, the effect of the recti muscles is to draw the eye *into* the orbit; that of the oblique muscles is to draw it out. Paralysis of the external rectus is easily determined. It is supplied by the sixth nerve. The third nerve supplies the superior, inferior, and internal rectus, the inferior oblique, the levator palpebræ superioris, the constrictor of the pupil, and the ciliary muscle. This third nerve divides in the orbit into an upper and a lower branch, the former supplying the superior rectus and the levator palpebræ superioris, the latter the internal rectus, the inferior rectus, the inferior oblique, the sphincter pupillæ, and ciliary muscle, and this latter also

sends a small branch to the superior oblique and external rectus. Paralysis of the third nerve may affect all the muscles completely or partially, or some may be completely paralyzed, whilst the rest are only partially affected, or there may be an isolated paralysis of one muscle. In complete paralysis of the third nerve the upper eyelid droops, and when it is lifted, and an object is moved before it in various directions, the eye fails to follow it in the upward, inward, and downward direction; but it can still move outward by means of the external rectus, and somewhat downward and outward by means of the superior oblique, although generally secondary contraction of the external rectus soon supervenes, and a marked divergent squint ensues with crossed diplopia. The pupil will be somewhat dilated and immovable. This branch of the sphincter pupillæ may, however, be paralyzed in advance of the paralysis of the whole nerve, and this I have frequently observed in cases of intracranial lesions. Finally, the eye will lose its power of accommodation. If the healthy eye is closed and the patient be directed to walk up to an object, he becomes vertiginous and rolls slightly, from the mental illusion as to the position of the object. There is also generally some protrusion of the eyeball on account of the paralysis of the three recti muscles. Marked ptosis is also generally present, and by relaxing the orbicularis and contracting the frontalis, the upper eyelid can still be slightly lifted. In paralysis of the internal rectus, we will say of the left eye, both eyes can follow an object nearly to the middle line, but beyond this the left eye will lag behind, giving rise to a divergent squint. In complete paralysis of the left internal rectus a rotatory, zigzag movement inward is observed when the left eye is moved inward, and this is due to the action of the superior and inferior rectus. When the superior rectus, say of the left eye, is paralyzed, the movements of the eye above the horizontal line will cause the left eye to lag behind, and this deviation will increase proportionately to the height of the body which is being moved, and there will also be a divergent squint due to the unantagonized action outward of the inferior oblique. If the right eye is covered, and the patient looks with the left eye at an object held slightly in the upper half of the visual field, the left eye will move upward and inward, according to the amount of paralysis, showing that it had before deviated downward and outward, and the covered eye will simultaneously make a considerably greater associated movement upward and inward. The patient will carry his head thrown back, so as to make as much use as possible of the lower half of the visual field, and in endeavoring to hit an object will strike too high. In paralysis of the inferior rectus the symptoms are just the opposite of those of paralysis of the superior rectus. Paralysis of the inferior oblique is very rare, and the symptoms are the opposite of those that obtain in paralysis of the superior oblique. Paralysis of the superior oblique requires some care to detect, and is often overlooked. Let us suppose that the left superior oblique has been paralyzed. The patient would see objects in the lower half of the field double and irregular in outline, but above the horizontal median line there would be no diplopia. If a body is held in the

horizontal median line, or a little below it, there would be a slight deviation of the left eye upwardly and inwardly, becoming more marked the more the object is moved into the lower half of the field, especially to the right. The left eye also moves distinctly downward and outward when the right eye is closed, but it must be remembered that this can only be perceived with a little care. In some cases a still more careful test should be made in paralysis of the superior oblique in order to determine the differences in the double images, but this is a matter for which reference must be made to the special text-books. A secondary contraction of the inferior oblique often supervenes after paralysis of the superior oblique has existed for some time.

Nystagmus is a spasmodic affection of the muscles of the eye, and consists in an oscillation of the eyeballs, generally horizontal but occasionally rotatory, and in some rare instances vertical. Spasm of the orbicularis palpebrarum, or blepharospasm, varies from a moderate temporary twitching to a firm pressure of the eyelids together, so that it may be impossible for the patient or the physician to open them even to a slight degree, and if this attempt is forcibly made, it may be very painful and cause convulsion.

Conjugate deviations of the eyes consist of simultaneous movement of both eyes to one side.

Strabismus is that condition of the eye in which the patient is unable to bring both visual lines to bear simultaneously upon one point. The inward strabismus is called convergent strabismus; the outward, divergent; the upward, sursumvergens; the downward, deorsumvergens. Convergent and divergent strabismus are usually due to errors of refraction, the former to hypermetropia, the latter to myopia, and therefore they are of little interest to neurologists.

That condition of asthenopia of the ocular muscles which is also known as insufficiency of the recti muscles has played a great *rôle* during the last few years in this country as the supposed cause of almost every affection of the nervous system from neuralgia up to general paresis, and it therefore deserves some little attention. It is a very common affection, and distresses the patient by causing the eyes to feel warm, uncomfortable, and strained in working or reading, so that the print becomes obscure, the letters run together, and there is often some pain through the temple, or forehead, or vertex, or an aggravation of existing neuralgic trouble of the trigeminus, and most especially of a tendency to migraine (*vide* Chapter IX., "Migraine"). In this condition vision and accommodation are good, and there is a normal appearance of the eyes. Direct the patient to look steadily with both eyes at an object; gradually bring this nearer to the eye, and when the object comes within about six inches of the patient, one eye becomes unsteady and wavering in its fixation, and then deviates in the direction of the weaker muscle, either gradually and slowly, or suddenly and spasmodically. When one eye is covered with the hand the same deviation occurs. If a prism is held with its base upward or downward so as to produce diplopia, this deviation, when dependent on a weak externus or internus, will

also be shown. It must be remembered, however, that the external rectus is able to overcome a prism ten, fourteen, or eighteen degrees for distance. In some cases of very latent insufficiency the eyes should be tested in a room which is moderately darkened; a lighted candle should be placed about twenty feet from the patient; this the patient fixes, then a red glass is put over one eye, then over the other eye a prism of five degrees, base upward. Two flames will then be seen. If the red glass is placed over the right eye and stands directly over the white of the eye, there may be an equilibrium of ocular movement; but if the red light go to the left, there will be insufficient adduction, and if it go to the right, insufficient abduction. If the lights, however, stand vertically, the candle should be moved several feet to the right or to the left, or the patient should be made to turn his head far to the right and then to the left, still regarding the flame; and in these lateral positions lack of perpendicularity will detect the faulty muscle. If the abductive power is more than eight degrees, insufficient adduction may be suspected, and this should be tested for by prisms with the base outward, increasing the strength of these prisms until the patient is no longer able to fuse the images. But as these tests should not be undertaken without proper correction of any error of refraction, the thorough method cannot be described in a book of this kind, and reference must be had to treatises upon the eye.

FIG. 94.

Atrophy of the disk from spinal disease. (From NETTLESHIP.)

FIG. 95.

Atrophy of the optic disk after papillitis. (From NETTLESHIP.)

An ophthalmoscopic examination of the eye is of great importance in many cases of nervous diseases, whether of the cord, brain, or peripheral nerves. This is a matter which belongs to the specialty of eye diseases more particularly, and for an explanation of the method of using the ophthalmoscope reference must be had to treatises upon the eye. Every neurologist should be fairly expert in the use of this instrument, sufficiently so, at least, to be able to distinguish the types of retinal lesion, such as ordinary retinitis, albuminuric retinitis, choked disk, and atrophy of the optic nerve. Although there is not sufficient space at my disposal to go into the minutiæ of these types, I have yet thought it would be of great practical value to insert the

accompanying illustrations. Fig. 94 represents an atrophy of the disk from spinal disease. Fig. 95 illustrates an atrophy of the disk after papillitis, the upper and lower margins being still hazy, the veins still

FIG. 96.

Albuminuric retinitis due to fatty degeneration. (From AYRE.)

FIG. 97.

Apoplectic retinitis of renal disease. (From AYRE.)

tortuous, but the arteries nearly normal, and there is disturbance of choroidal pigment at the inner and outer border; although the disk is not represented quite white enough. Fig. 96 portrays an albuminuric retinitis due to a fatty degeneration of the fibrous tissue of the retina, especially localized in the radiating fibres of the retina around the origin of the yellow spot, whilst in Fig. 97 is seen an apoplectic form of neuro-retinitis sometimes seen with renal disease.

Testing for hemianopsia is readily done, but the distinction between hemiopia and hemianopsia should not be forgotten. A rough test is for the physician to hold up his forefinger before one eye of the patient, the other eye being closed by the patient's fingers gently pressed upon the lids. A white object, such as a handkerchief or a piece of chalk, is now brought from the side toward the examiner's finger, and the patient is asked if he can see this object whilst he is looking straight at the finger. If he can, this lateral field of vision is good. In this manner the upper, lower, lateral, and median fields of vision can be tested. For cases that are to be recorded, so-called perimeters are useful in exactly marking off the defects of the field of vision, and charts for recording these measurements are sold at all the instrument-stores.

HEARING.

The hearing should be tested in order to determine the capacity for hearing, as well as for that symptom which is known as mental deafness or inability to understand the meaning of what is heard.

The tests for simple hearing are ordinary conversation, the tick of a watch, and the tuning-fork. Really the best test is that by means of conversation; but as this is apt to consume too much time in the hurry of office-work, the next best means would be by some such test-sentences as those suggested by Buck, two of which are:

> Pour oil on the waters of Lake Erie;
> All hail! thou hero of fourteen wars.

Each of these sentences contains eight long vowel sounds and is equally free from non-resonant consonant or short vowel sounds. The next best test is by means of a watch, although experience has taught me again and again that this must be corrected by the test of sentences. The watch should be placed at a distance where its ticking cannot be heard by the patient, and then gradually approached to where the ticks can be so heard, and the latter may be regarded as the farthest point of distinct hearing. The objection to first placing the watch close to the ear and then removing it to a distance is that when a sound is once perceived, it is easy for the patient to imagine that he hears it beyond where he really does perceive its sound. The ear that is not undergoing examination should be closed, of course. There is no hard-and-fast rule as to exactly the distance at which a watch can be heard by a normal ear, because watches vary greatly in the loudness of their sound, most of them being heard best immediately

after being wound, and also when held so that the physician's hand covers the back, or when held by the patient. But four feet from the ear may be taken as the proximate normal distance, although, as I must reiterate, there are many exceptions to this rule. Some measuring-instrument, as a tape-or a yard-measure, should be employed in order to determine the distance from the ear to the watch.

The tuning-fork is generally employed in order to determine whether there is disease of the middle or internal ear, and also for testing hearing, but it has not been popular with aural surgeons. If we close our ears and speak, the sound seems to be confined to the cranial cavity, so to say, and its reflection is to a certain extent prevented by the occlusion of the external auditory canal. If the auditory nerve be normal, and if there be cerumen in one auditory canal, or a thickening of the mucous membrane of the tympanic cavity, there will be a condition similar to closure of the external meatus of a healthy ear by the finger, and the vibration of the tuning-fork placed upon the bones of the head will be heard most distinctly by the affected ear, although a conclusion cannot be reached in this way if both ears are equally implicated. If there is disease of the auditory nerve, either from a direct lesion of it, or from disease which has extended from the middle ear to the internal ear, no such marked difference will be noticed when the external meatus is closed. If there is no serious lesion of the auditory nerve, although the tick of the watch may not be heard, the vibrations of the tuning-fork placed upon the teeth, forehead, or mastoid process will be distinctly perceived. If there is serious lesion of the auditory nerve, causing absolute deafness, these vibrations will not be perceptible in the head. Some persons who are not trained to observation will maintain that they hear the tuning-fork better from the better ear, because they believe that they ought to do so, but it is much more reliable to take the statement of a patient as to whether the tuning-fork is heard more distinctly and longer when its vibrations are conducted through the ear or through the bones of the head than it is to rely upon his statement as to which side he hears it better on. The large tuning-forks of the note C should always be employed. If hearing is impaired, and the conduction of the vibrations of the tuning-fork is better through air than through bone, there is disease of the auditory nerve, and this may be either primary, or secondary to disease of the middle ear. If the conduction of the vibration of the tuning-fork through bone is increased, and lasts longer than aërial conduction, there is disease of the middle or external ear. In order to test the conduction of the ear and the bone-conduction relatively, the tuning-fork should be struck on the knee of the examiner, and then held in front of the meatus to test the vibrations through the air, then again set in vibration and its handle placed on the mastoid process near the centre to test the bone-conduction. The explanation of the fact that the tuning-fork is heard best through the bone when there is disease of the middle ear is because the increase of diseased tissue increases the capacity of resonance of the parts, and when there is disease of the auditory nerve the vibrations pass through the ex-

ternal auditory canal, the tympanic cavity, and the fenestra ovalis, which constitute the best channels of conduction.

In dealing with the symptoms of Ménière's disease (*vide* Chap. XV., "Ménière's Disease") the neurologist has often to make a diagnosis between disease of the middle ear and disease of the labyrinth. This can be done mainly by attention to the following facts:

Air-conduction is best through a normal ear, so that such conduction is heard three or four times better than bone-conduction. In disease of the middle ear, however, bone-conduction is greater than normal, and air-conduction is less, so that the relations of air-conduction to bone-conduction are precisely the opposite to what is the case with the normal ear. When there is disease of the internal ear the bone-conduction is weakened, and the air-conduction is also weakened, but it frequently happens that the relation between the bone-conduction and the air-conduction is greatly in favor of the latter; *i. e.*, air-conduction is better than bone-conduction, although both are weakened. In mixed cases in which disease of the middle ear and disease of the labyrinth are combined there are: 1, the disturbed relation between air- and bone-conduction which is usually found in middle-ear disease; and 2, the bone-conduction is weaker than normal.

Pomeroy subtly calls attention to the fact that when the normal ear is stopped with the finger, and the bone-conduction is therefore at its maximum for such a normal ear, an incautious examiner might compare it with the bone-conduction of an ear suspected of diminished bone-conduction, but which had not been closed by the finger. Pomeroy also states that while the rule is that a normal ear stopped with the finger will have its bone-conduction at a maximum, yet in some diseases of the middle ear, with the meatus stopped, the bone-conduction is somewhat better than normal, although these are somewhat exceptional cases.

The symptom of word-deafness is that in which, as has been explained, the patient hears distinctly what is said, but fails to understand the meaning of it, and it is due to a lesion of the posterior portions of the first and second temporal convolutions, or to the conducting-strands leading from them down to the periphery.

SMELL.

In testing smell, distinction should be made between the impression made upon the fibres of the trigeminus that supply the nostrils and that made upon the olfactory fibres. The former can be tested by pungent odors, such as ammonia, alcohol, etc., whilst the latter should only be tried by the pure odors, of which the best are the odor of violet, oil of cloves, and musk. One nostril should be gently closed by the physician's hand by pressure inward until the septum is gently felt, and then the other should be tested by means of the odorous substance, the nature of which should not be made known beforehand to the patient. The difficulty in this test is, that the olfactory nerves in the human being soon weary, so that a test cannot

be repeated more than a few times. A careful examination of the anterior and posterior nares should always be made in order to see that any loss of smell is not due to some lesion of the mucous membrane or the bones.

TASTE.

In testing the sense of taste it should be remembered that the gustatory filaments of the tongue come from two different nerves, the anterior half being supplied by the lingual nerve issuing from the chorda tympani, whilst the posterior half is supplied by the glosso-pharyngeal nerve. The test should be made by means of a bitter, such as quinine; by a sweet, such as sugar; by a sour, such as vinegar; and by a salt such as common salt, and it is best to test successively with sugar, then salt, then vinegar, and last of all the quinine, as the bitterness of the quinine will blunt the subsequent gustatory impressions if it be used first. The patient should be made to protrude the tongue, when first one posterior half and then the other should be tested, and after this the anterior half, the patient having been instructed beforehand not to draw in the tongue and allow it to come in contact with the roof of the mouth, but to indicate whether he tastes the substance by shaking the head for a negative and pointing the finger for an affirmative. In this way the sense of taste can usually be localized in an intelligent person. The electrical examination of the sense of taste is of very little value, because the sharp and acrid taste of the electric current is not so fine a test as the different substances that have been mentioned.

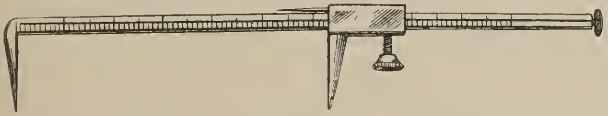
There has been great discussion among physiologists as to whether the senses of touch, pain, temperature, muscular sense, and weight are really distinct, or whether they are not variations of feeling in the same nerves. However this may be, it is certain that clinically we constantly find one or more of these senses disturbed when others are intact, and it has even been claimed in late years that the loss of the temperature-sense especially is a diagnostic mark of syringo-myelitis. I have no hesitation, therefore, in discussing them separately from the clinical point of view.

TOUCH, OR TACT.

The best method of testing the sense of touch or tact is to see if the patient can distinguish between different substances lightly brought in contact with the skin, such as a piece of cotton or a silk handkerchief and the touch of the physician's finger. In order to do this in the fingers, for example, the patient's eyes should be closed and something held in front of them, when the finger-tips should be lightly touched, first with the cotton or handkerchief, then with the physician's own finger-tips, and the patient should tell which is which. This test, simple as it may seem, yet requires a certain amount of care. If a test is first made with the cotton, for instance, and then

with the finger, and this rotation is regularly kept up, first cotton and then finger, the patient may become accustomed to it and anticipate rather than feel. The best way is first to take the patient's hand, and then, not touching it at all with the cotton or the handkerchief, ask him what he feels. If he says that he feels either the handkerchief or the cotton, he should be at once informed that he has not been touched at all, so that in this way he will be made more alert, and the examination should not actually be made until the patient is really seen to be informing the physician of what he feels rather than of what he imagines. The cotton or the finger should then be brought very lightly in contact with the finger so as to test the degree of sensation, whether there is impairment of it or whether there is increase of it. The examiner should be familiar with the degree of sensitiveness of different parts of the skin, and this can only be acquired by a certain experience. Slight differences cannot be determined except after repeated examinations, because the sensitiveness of touch varies very greatly from one individual to another, and also in the same person according to varying corporeal conditions. But no test of the sensation of touch can be properly made if the skin is cold, and in all examinations of the feet or hands they should be carefully felt, and if cold, they should be gently warmed before the test is made. It is a familiar fact that typesetters will make many mistakes in setting type if the temperature of the room goes below a certain point, and this is also true of typewriting. The instrument known as the æthesiometer, invented by Sieveking, is, as the following figure shows,

FIG. 98.



Æsthesiometer.

a movable slide upon a horizontal bar measured off into inches, or metres and millimetres. By means of this the distance to which the two points need to be separated in order to be felt as two points can be determined, and this distance has been found to vary in the different areas of the skin, as is shown in the following table of Weber:

Tip of the tongue, 1 millimetre.
Finger-tips, 2 millimetres.
Lips, 3 millimetres.
Dorsal surface of the first and second phalanges and the median surface of the fingers, 6 millimetres.
Tip of the nose, 7 millimetres.
Thenar and hypothenar areas, 8 millimetres.
Chin, 9 millimetres.
Tip of the great toe, the cheeks, the eyelids, 12 millimetres.
The glabella, 13 millimetres.
Heel, 22 millimetres.
Back of hands, 30 millimetres.

The neck, 35 millimetres.
Forearm, leg, and back of the feet, 40 millimetres.
Back of the trunk, 60 to 80 millimetres.
The upper arm and thigh, 80 millimetres.

The difficulty about the æsthesiometer is practically that unintelligent people, such as the mass of those who come to our clinics and hospitals, are very apt to become confused, as a little experience will demonstrate, and the differences are very great from one examination to another. This is not surprising when we reflect that this instrument appeals to a peculiar form of tactile sensation which is unfamiliar to the ordinary human being; and for this reason, at the first examination I prefer the test for the tactile sense spoken of above, employing the æsthesiometer only in order to determine with approximate accuracy the variations which may occur in the course of treatment. This table of Weber's, however, must be taken with due allowance, as these distances vary very greatly from individual to individual and in a given individual on different days, because of the varying bodily conditions and because of the unfamiliarity of the instrument. Variations, therefore, should not be regarded as abnormal unless they are decided; thus, if the instrument had to be separated four millimetres in order to be felt at the finger-tips, this would be abnormal, or if on the thigh they were felt at forty instead of eighty, this would be abnormal, but a variation of one-half or one millimetre in the finger-tips or of one to two millimetres in the thigh should not be hastily interpreted. In testing for medico-legal purposes, or where there is any suspicion of malingering, it may be necessary to resort to indirect tests of the tactile sense. Thus, if a person claims that he has lost the sense of touch and cannot feel the difference between silk or cotton and the finger, or that he cannot feel the points of the æsthesiometer widely separated beyond what should be the normal point, the subject may be quietly changed and an opportunity sought for during the examination to touch lightly with the finger the part that is claimed to be anæsthetic, when the patient's attention is attracted to something else; and then some evidence of the touch having been felt may be obtained. In this way I have often detected malingering; in one case lightly touching the patient's neck when her back was toward me, whereupon she turned her head sharply around, and in another case by lightly tapping the man's hand with mine as his head was directed away from me, when the immediate quick turn of the head and the inquiring look made it evident that the touch had been accurately felt. A quick-minded and alert examiner can almost invariably throw a malingerer off his guard by this method.

PAIN.

The best test of the sense of pain is by the hidden needle, or *aiguë caché*. This is a needle within a hollow cylinder, and to the upper end of it is attached a spring, which in its turn is attached to a spiral thread running up and down through a slot by the working of

a little circular cap. The extent to which the needle can be made to protrude can be determined beforehand. This instrument is placed upon the patient's skin, very few people knowing what it is, and the needle is pressed quickly down and inserted into the skin, so that it is a beautiful test—as perfect as can be devised—of the pure sense of pain, without any anticipation upon the patient's part. Unfortunately, however, it will sometimes happen in this day of damage suits against corporations that individuals are acquainted with the instrument, and it requires very little fortitude to stand the prick of this pin without any manifestation of pain, which is not surprising when we reflect how readily patients become accustomed to the insertion of hypodermic needles for inches under the skin, whilst most schoolboys are familiar with the schoolboy trick of doubling up the leg under the thigh, then drawing the skin over the knee tense, and suddenly jabbing in a pin without expressing any feeling of pain. In any doubtful case it is well, therefore, to make use of some other test than the hidden needle, and this can always be done by means of an electric battery. The battery should be well tested beforehand, in order to ascertain that a powerful current can be turned on at will. Small electrodes of copper (Figs. 72 to 76) should be used, and should be separated only a few inches. A gentle current should be first used, and if the patient claims that any pain is felt, a feint should be made for a minute or two of testing with this gentle current, and then, after the examiner has apparently satisfied himself that no pain is felt and the patient is thrown off his guard, a powerful current should be suddenly turned on. This, however, will not be conclusive, because the patient may really have an impairment of the sense of pain and yet not an absolute loss of it, so that a lighter current might really not be felt, whilst the most powerful current would be. However, this primary procedure has the advantage in many cases of causing the patient to break down at once and admit that he has been shamming. If he does not do this, the examination should be resumed, and starting with the moderate current, it should be gradually increased until it is determined relatively what current is really felt. By the time this second examination is in process a shamming patient has usually become so demoralized that any further shamming will be easily recognized, whilst a partial anæsthesia in a truthful person will be determined with equal readiness. Marked degrees of anæsthesia, however, can often be determined by the needle, because it often happens that such anæsthesia is accompanied by an ischæmia of the anæsthetic part, which is demonstrated by the sluggish flow of blood following a deep needle-prick, and this blood is usually venous in character. Moreover, the needle-prick can be made to serve a good purpose by being used upon the anæsthetic part when the patient's attention is attracted to other matters.

MUSCULAR SENSE.

The muscular sense should be tested by bending a joint or a limb with the patient's eyes blindfolded, and then having the patient tell

what movement is made. In the slighter degrees of impairment of the muscular sense the small joints can be moved without the patient knowing it, whilst the larger joints cannot, and it is a high degree of anæsthesia that permits of the larger joints or a whole limb being moved without the patient being aware of it. If anyone has a doubt of the difference between the muscular and tactile sense, they have only to have an experience, as I have daily in my clinics and lectures, of absolute impairment of tactile sense and preservation of muscular sense, and *vice versa*.

TEMPERATURE-SENSE.

The simplest test of the temperature-sense for clinical purposes is to take three tumblers or test-tubes of water, one cold, one luke-warm, and one hot, and have the patient tell which is which with blindfolded eyes. Care should be taken, however, that the eyes are blindfolded before the water is brought into the patient's presence, otherwise quick-minded individuals will see the slight steam from the water or notice the greater transparency of perfectly cold water. A more accurate test can be made by a series of test-tubes with water at different degrees of temperature as tested by an ordinary axillary thermometer. In the Salpêtrière, in Paris, the instrument shown in Fig. 99 is used. It is difficult to test the temperature-sense in malingerers, but I have sometimes succeeded in exposing individuals who claimed that they could not feel differences of temperature by suddenly spilling some hot water upon the hand when their attention was directed to other subjects.

Fig. 99

Blocq's thermo-æsthesiometer.

WEIGHT.

The sense of weight has not been shown to be clinically of much importance, and we know very little about the significance of variations in it. It can be tested by superposition of varying weights. The hand should be well supported by being laid gently upon a table, and the weights should be superimposed at regular intervals as measured by the second-hand of a watch.

THE TENDON-REFLEXES.

The tendon-reflex consists of the muscular jerk which results from mechanical irritation of the tendon of the muscle, and it is something entirely distinct from the muscle-reflex, which is due, as the name indicates, to mechanical irritation of the muscle itself. All muscles have their tendon-reflexes, of course, so that in order to speak of them fully we should catalogue every muscle in the body. As a matter of fact, however, there are only a few muscles which are of

sufficient size and anatomically so situated as to enable us to play upon their tendons and obtain a jerk of the muscle in health, although in many diseased muscles we can obtain reflexes which are impossible in the normal condition. The tendon-reflexes which are of clinical importance are the patellar tendon-reflex or knee jerk, the triceps tendon-reflex or elbow-jerk, the jaw-jerk or tendon-reflex of the masseter muscles, and the tendon-reflex of the leg-muscles, or the so-called ankle-clonus. The most important of these is the patellar tendon-reflex—the knee-jerk, or the tendon-reflex *par excellence.* This can be evoked in several ways. The patient can be made to cross one knee over the other, and whilst it is thus hanging percussion can be made upon the tendon of the quadriceps extensor, as it is stretched over the flat surface of the tibia. The best means of percussion, I think, is that which I am accustomed to use in my clinics. The middle finger of the right hand is extended, the index-finger placed back of it so as to support the distal phalanx, and the thumb is put in front of it in a corresponding situation. By this means a strong and delicate percussion-hammer of Nature's making is ready at hand, with which one can tap strongly or tap lightly, so as to bring out a feeble tendon-reflex or show the great irritability of a very much exaggerated one. A percussion-hammer may also be made use of, or the tendon may be tapped with the side of the hand, but none of these methods is equal to that just described in precision and facility of range from a light to a heavy blow. This knee-jerk when feeble can often be made more forcible by what is called the method of "reinforcement." Jendrassik does this by having the patient lock the fingers of each hand and pull them forcibly apart; and Mitchell and Lewis have shown that the same effect can be obtained not only by powerful muscular acts, but even by winking, speaking, coughing, laughing, pulling the hair, touching the skin with iron or something hot, pinching, or electrical excitation upon any part of the body. The jerk forward of the foot should be carefully observed for two kinds of phenomena: first, the amount of muscular movement; and, second, the spastic element evident in the muscles. Thus, a simple increased jerk may be obtained, or a spastic jerk, or a combination of the two. Clinical observers have not, in my opinion, by any means laid the stress upon these two kinds of phenomena that they deserve. It may often be extremely difficult to determine what is a normal knee-jerk forward of the foot, because the range of the normal is so great; but a spastic element, a quick jerky twitch forward, is always abnormal, even when the muscular range of the movement is small. The tendon-reflex varies very greatly in health. Thus, among 200 healthy medical students whom I examined, in 5 I found the tendon-reflex absent, in 180 of an approximately normal range, and in 15 apparently exaggerated, but in none of these did I find any spasmodic element.

The jaw-jerk is tested in the following manner: The patient is made to open the jaw, and then the masseter muscles are tapped with

a percussion-hammer or the finger held in the way just described. This tendon-reflex is seen only in disease.

The foot- or ankle-clonus (also formerly called spinal epilepsy by Brown-Séquard) is evoked in one of two ways: 1, the patient is made to extend his leg and thigh, when the physician should grasp the back part of the calf with the left hand and the anterior portion of the sole of the foot with the right hand. The patient should then be instructed to remove his voluntary control from the muscles of the foot, and whether this has been done should be determined by gently flexing the foot upon the leg, as the same difficulty is often met with in uneducated people in this regard that will be spoken of in describing the test for slight contracture. When it is certain that the patient has "let go" of his foot, the latter should be quickly and forcibly flexed upon the leg, when any exaggerated tendon-reflex will be made evident to the examiner's hand by a series of throb-like extensions of the foot, which will pass into a continuous coarse tremor if flexion of the foot is firmly maintained; 2, the patient should be seated upon a stool and then instructed suddenly to tap the toes of one foot firmly upon the floor, keeping the heel well elevated, when an exaggerated tendon-reflex may be shown by a series of rhythmical contractions of the muscles of the legs, constituting a coarse tremor also In some cases the ankle-clonus can be evoked by only one of these methods, but in others both methods will produce it, and I have frequently been able to call it forth by one method one day and then failed to obtain it the next day in the same patient except by the other method, and perhaps upon a further day failed to evoke it in this individual by either method.

It is still a matter of doubt as to whether the knee-jerk is a direct muscular process or a spinal reflex act. It has been observed that the duration of the reaction-period is shorter than that of spinal reflexes. Nevertheless, section of either the sensory or motor spinal nerve-roots abolishes the knee-jerk, so that it is apparently dependent in some way upon the spinal cord. This I have verified in my own experiments, and I have also found that such a slight inhibition as lifting the crural nerve lightly upon a string abolishes the knee-jerk of a rabbit, and that this knee-jerk immediately reappears when the string is withdrawn. Another theory has been that nervous excitations continuously transmitted from the spinal cord to the muscles maintain the so-called muscular tonus, but Lombard could find no proof that muscular tonus was continuous, or that the irritability of a muscle to a mechanical stimulus was dependent on its tension. H. C. Wood, refining the matter still further, has suggested that the irritability of the nerve in the sarcolemma of the muscle might be dependent on the spinal cord, and that the knee-jerk was dependent upon the irritability of these nerves, so that it might not be a reflex act, and yet not be dependent upon muscular tonus. But, as Bowditch pointed out, the irritability of the nerve is not lessened for some time after section, although the knee-jerk is lost immediately. As to how the "reinforcement" method increases the knee-jerk we are equally in the dark. There are two theories upon

the subject : one is that the inhibitory action of the cerebrum is cut off from the parts below through the nervous energy being diverted to other paths by the voluntary act of reinforcement of such muscular efforts as coughing, singing, etc. ; and the other theory is that an excess of energy is expended in these acts of reinforcement which exalts the excitability of other centres. Neither theory, however, has been proved or disproved. The knee-jerk varies in healthy individuals to a considerable extent under the influence of fatigue, sleep, changes in the weather, emotions, different sensory impressions, or mental effort, and, as we shall have occasion to see in the various chapters of this book, alters greatly from time to time in cases of disease.

Gowers believes that the essential condition to all these tendon-reflexes is that of passive tension of the muscle, not necessarily of the tendon, and he, therefore, suggests for them the name of *myotatic* contractions (from τατικὸς, extended).

CUTANEOUS REFLEXES.

The chief cutaneous reflexes are the cremaster, the plantar, the mammary, and the abdominal.

The cremaster reflex can be tested by having the patient stand up, so that the testicles and the penis hang, and then scratching gently with the nail or with a blunt instrument along the inner side of the thigh, when the testicle will be seen to be drawn up. This reflex can generally be evoked within an area covering the inner side of the thigh, about to the middle line in front and about to the edge of the curve of the thigh inwardly ; but when it is exaggerated it can be elicited by excitation of farther-lying portions of the thigh, even the back of it, or by irritation of the abdominal cutaneous surface. It has not yet been proven to be a sign of much diagnostic importance, but I have seen it notably exaggerated in many cases of reflex spastic symptoms, pain from stricture, disease of the seminal vesicles, and occasionally from disease of the prostate gland.

The plantar reflex is the reflex contraction provoked by tickling the sole of the foot, the muscles of the leg, or even of the thigh. In some cases mere tickling will not call forth this reflex, but it will have to be elicited by pricking with a needle, or by the faradic brush, or by a sponge dipped in hot water, or even by putting the limb into hot water.

The mammary reflex is obtained by tickling the breast, when the nipples are erected by the contraction of the muscular fibres.

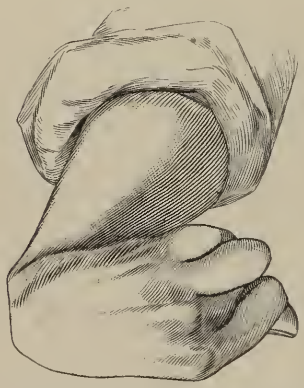
The abdominal reflex is the contraction of the muscles of the abdominal wall when the skin over the abdomen is irritated by the finger-nail, or by a blunt object, or with something cold or something hot. The upper part of this reflex is known as the epigastric reflex.

CONTRACTURE.

Contracture is a condition of the muscles which differs radically from contraction. In the latter the muscle is locked, as by fixation

of a joint or by callus. In the former the muscle is contracted, but the contracture is not fixed, and can be overcome by gently pressing upon the muscle, the contracting muscle giving to the examiner's hand the sensation as of wax gradually yielding. This condition generally, almost invariably, affects the flexor muscles, and varies in degree from the slight contraction that can only be evoked by careful examination to the pronounced degree in which the muscles are drawn into positions of great deformity, as in Figs. 100 and 101. When the condition is very marked there is no difficulty whatever in detecting it, but slight degrees of it require some care to elicit, and I have for years been in the habit of employing the following methods in my clinics: We will suppose that the muscles which we are examining are those of the lower extremity. The patient is instructed to extend the leg and then to give it up passively to the examiner. Many uneducated people find it very hard to remove ab-

FIG. 100.



Contracture of the hand in a case of hemiplegia from hemorrhage into the internal capsule. Dorsal view.

FIG. 101.

Contracture of the hand in a case of hemiplegia from hemorrhage into the internal capsule. Palmar view.

solutely their voluntary control of a set of muscles, and therefore the leg must be bent at the knee several times in order to determine that the patient has allowed it to extend and flex with perfect flaccidity. When this has been satisfactorily determined, the leg is gently extended upon the thigh, one of the examiner's hands grasps it above the ankle, whilst the other hand of the examiner is placed upon the thigh, and then a sudden movement of flexion of the leg should be made. If there is any slight contracture of the muscles, the examiner's hand will become aware of a sudden movement of resistance in the leg, like a muscular "click," and the hand is thrown back to a varying degree, usually sufficient, even in the slightest cases, to be perceptible to the eyes of the class. A very little experience will enable a person to make a quick distinction between this involuntary muscular resistance or click of the leg whose muscles are contractured and the resistance which may be made voluntarily by the patient, and I never have any difficulty in teaching my class to make this

distinction in one lecture. I believe that this method of mine is of very great value in determining slight degrees of contracture, and in several cases I have been able by it to solve the question as to whether there was any malingering. When the contracture is marked, this sensation of muscular resistance or click is very distinct, and the examiner's hand will be thrown back several inches as he attempts to flex the leg. The great mistake made by most of my students is that the movement of flexion is not quick enough, as a slow movement of flexion will not evoke this muscular click. The phenomenon is evidently dependent upon the increased muscular or tendinous excitability—I am not certain which it is, although I believe that it is the latter, as in all of these cases the tendon-reflexes are exaggerated, while the muscular excitability is not always above normal. Indeed, it may be laid down as a rule that tendon-reflexes, muscular contracture, and hasty micturition are symptoms that go together; and in some cases of paraplegia, especially when due to spinal syphilis, these three symptoms become annoying to the highest degree, so that the urine must be passed at very frequent intervals during day and night, the legs are painfully flexed upon the abdomen by the contracture, and, as I have seen in several instances, the tendon-reflexes are so enormously exaggerated that the least movement with the bedclothes, or a loud sound, or a tap upon the limb, will cause the legs to be drawn up with a series of twitches and jerks, whilst, if the patient is placed in a chair, the legs will be seen in constant but irregular motion, sometimes in movements of flexion upon the abdomen, first upon one side and then upon the other, then in rigid, bar-like extensions of the legs upon the thigh, or in flexion of the legs upon the thigh, until life is made more than miserable.

PARADOXICAL MUSCULAR CONTRACTION.

This phenomenon was first described by Westphal as a slow tonic contraction in a muscle which has been suddenly relaxed. It is best seen in the tibialis anticus, and can be evoked by grasping the foot and passively flexing it on the leg. It has also been observed in the extensors of the toes, occasionally in the flexors of the knees, rarely in the arm-muscles. It is not accompanied by muscular rigidity or by exaggerated reflex of the muscle itself, although there has sometimes been diminution of the latter. It is as yet a clinical curiosity, and its cause is unknown.

MUSCULAR INCO-ORDINATION.

Ataxia is that condition of muscular inco-ordination in which precise combined movements of the muscles cannot be performed with the normal exactness, although there is no loss of muscular strength. Muscular inco-ordination, or ataxia, can be detected in several ways. The best test of the lower extremities is to have the patient walk along a straight line, such as a crack in the floor, the edge of a rug, or a straight line running through the carpet, and do this with the eyes

closed, so as to be entirely dependent upon the sensations coming through the sensory nerves of the lower extremities. A normal person can walk this straight line with steadiness and ease, whereas a person who has ataxia will stagger slightly, occasionally throw one leg out to maintain the equilibrium, or in the greater degrees be totally unable to hold himself erect. Ataxia of the upper extremities can be tested by having the patient shut the eyes and then swing one arm up from the side of the body with a long swinging movement, so as to touch the tip of the nose with the tip of the forefinger, when inability to co-ordinate the muscular movements will be seen by a failure to bring the finger to the nose, the finger either hitting the side of the nose, or wavering as the nose is approached, or, in the severe cases, hitting only the side of the head, or the ear, or failing altogether to perform any purposive movement toward the nose. This ataxia of the muscles of the extremities is called *motor ataxia.* The ataxia which a person has in the trunk in the sitting position is called *static ataxia*, and it can be seen by the swaying and inability to maintain steadily upright the trunk of the person who is seated. Some authors speak of the so-called *Romberg symptom* as an evidence of ataxia. When this is present, the patient, standing with the eyes closed and the feet closely approximated, sways to one side or the other, or backward and forward. I do not regard this, however, as a sign of muscular inco-ordination or pure ataxia, but rather a disturbance of equilibrium due to so many different diseases of the central and peripheral nervous system that its diagnostic value is very small. Other authors, again, speak of the standing upon one foot with the other raised as a good test of ataxia, but there are many perfectly healthy people who cannot do this well, and it is a very unreliable and clumsy test.

In these tests which I have spoken of for ataxia, great care must be exercised to have the patient's eyes closed, and not to have him touch with the hand any neighboring object or any person, because, as I shall show when treating of locomotor ataxia, a patient who may have so pronounced an inco-ordination of muscles as to be in danger of breaking his neck in attempting to walk with the eyes closed may yet so walk with security and relative ease when he is simply allowed barely to touch his own finger to the finger of another person, as by this method he obtains, through the sense of touch and the muscular sense of his unaffected or slightly affected upper extremities, a guide to his movements which is lacking in his affected lower extremities.

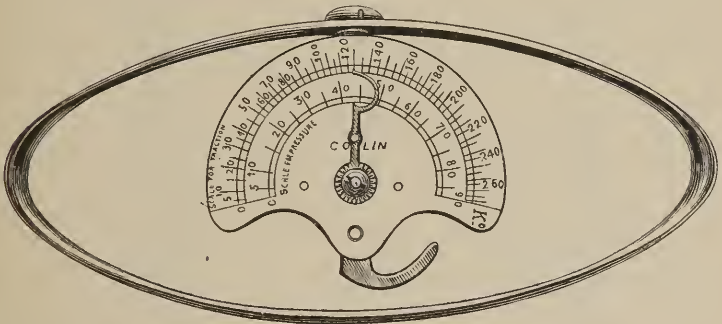
The causes of muscular inco-ordination are varied. It has been observed in lesions of the peripheral nerves, in locomotor ataxia, in lesions of the pons—especially of the lemniscus—in disease of the cerebellum and its peduncles, in affections of the cerebrum and also of the auditory nerve. It is probable, therefore, that ataxia may be produced by any defect in any of the nerves conducting the various kinds of sensation. We know, as has already been said, that a defect in the conduction of the waves of sound will produce it, as in lesions of the auditory nerve. We know that it is often seen in

those cases of peripheral neuritis in which there is only impairment of the sense of touch and pain. We know that it can be produced by lesions of the lemniscus, which is a sensory tract. We know that it can be produced in such a spinal disease as locomotor ataxis. We do not know, however, exactly which one of the four senses of tact, muscular sense, pain, and temperature, must be impaired in order to produce it, although it is very probable that it is never due to lesion of the temperature- or pain-sense. I am inclined to think that it is often due either to lesion of both the muscular sense and the sense of touch, or of the sense of touch alone. That it is not dependent upon loss of the muscular sense alone, I have demonstrated several times to my classes by exhibiting ataxic patients in whom the muscular sense was either normal or so slightly impaired as not to be able to account for the very pronounced ataxia, as well as other patients whose muscular sense was greatly affected and who yet had no ataxia.

MUSCULAR STRENGTH.

Muscular strength is to be tested, of course, by determining the muscular power of the different muscles. Fig. 102 represents an instrument for testing the strength of the muscles of the hands. By

FIG 102.



Dynamometer.

taking it into the palm and squeezing it, the strength in kilogrammes is indicated upon the lower scale, that for pressure; whilst locking a finger in each oval end and pulling will indicate the strength upon the scale for traction. The defect in this instrument is that the spring is variable in its elasticity and soon becomes weak, so that the measurements are not absolute. It is preferably used, therefore, to test the strength from time to time in a given case. The best means of testing the strength of the hand-grasp is, I think, the one which I have devised: I give the patient my hand in such a way that he cannot grasp it above the knuckles, so that he cannot hurt me unless he possess unusual strength, because my fingers roll over one another and prevent him from obtaining a firm hold. One soon becomes an

expert in hand-grasps if this method is used, for all civilized mankind is already familiar with the varying degrees of hand-pressures and their shades of meaning. Just as the gynecologist has an educated finger, so should the neurologist have an educated hand. I can readily detect slight variations of strength by my method.

There is no convenient instrument for testing and recording the muscular strength of the foot and leg; but, after all, in every case of muscular impairment, anatomical knowledge, beyond the possibility of any instrument, is needed to detect the individual muscles at fault. In most cases, too, the gait is of great value in diagnosis, for the drop of the toe in extensor paralysis, the flop of the foot in flexor impairment, the wrist-drop in lead-paralysis, and other characteristic deformities tell as much as an instrument or a careful examination of the muscles involved.

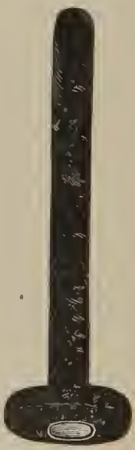
CEREBRAL THERMOMETRY.

In 1878 I presented to the American Neurological Association the results of an investigation of cerebral temperatures of 102 individuals. The places at which I placed the thermometers I denominated *stations*, and they were situated as follows: One on each side

FIG. 103.

Cerebral thermometer.

FIG. 104.



Cerebral thermometer with cover and pad.

somewhat back of and above the commencement of the external angular process of the frontal bone, the *frontal station;* one on each side just above the ear, the *parietal station;* and one on each side of the occiput, the *occipital station.* About over the fissure of Rolando —*i. e.*, about six and seven-eighths inches back from the furrow formed by the junction of the nasal bones with the nasal process of the frontal—was the *vertical station*, two on each side. I had made a series of surface-thermometers, great care being taken to see that they registered perfectly and that the flat bulbs were not sufficiently plia-

ble to have the registering scale of mercury altered more than one-half a degree by strong pressure. These thermometers were held in place by a strip of soft morocco running around the head and upward over the line of the great longitudinal fissure to the occiput, and perforated so as to permit the insertion of the thermometer at the requisite stations. In addition to this, each thermometer had around the bulb, sliding over the upright scale, a round padded cushion of silk, and over the upright scale was slipped a glove of silk, these precautions being taken to prevent the environing temperature from affecting the mercury. (Figs. 103 and 104.) The conclusions which I arrived at were these:

1. The average temperature of the left frontal station was 94° 36′, the right being 93° 71′.
2. The average temperature of the left parietal station was 94° 44′, the right being 93° 59′.
3. The average temperature of the left occipital station was 92° 66′, the right being 91° 94′.
4. The average temperature of the left side of the head was 93° 83′, the right being 92° 92′.
5. The average temperature of the whole head, exclusive of the vertex, was 93° 51′.
6. The average temperature of the motor region of the vertex was 91° 67′.
7. The average temperature of the whole head, inclusive of the vertex, was 92° 66′.
8. If there be an alteration of temperature at any of the lateral stations, this fact will justify a *suspicion* of abnormal change at that point.
9. If there be an alteration of temperature at any of the lateral stations of more than two degrees above or below the average of such station, this fact will constitute strong evidence of the existence at this station of abnormal change.
10. In proportion as the alteration of temperature at any individual station is increased or decreased beyond the figures just mentioned, in exact proportion will the strength of the evidence be increased, until, the maximum or minimum having been passed, the evidence will become almost conclusive.
11. If it should so happen that such elevation of temperature above the average should be at any lateral stations on the right, causing a rise at this point beyond the average temperature at the corresponding station on the left, this would strengthen the suspicion or the evidence.
12. These remarks apply with equal force to the average for the whole of either side, as well as to the average for the whole head.
13. It is necessary to the validity of these conclusions that the contemporaneous bodily temperature should be normal, or that there should be a marked disproportion between it and the cerebral temperatures.

I then put on record the first case of diagnosis of an intracranial lesion by cerebral thermometry—a glioma of the brain, in which the

clinical symptoms pointed to an implication of the base, but which I located at the lateral aspect of the cerebrum, and in which the autopsy confirmed my diagnosis. In this the tumor was found between the horizontal branch of the fissure of Sylvius and the first temporal fissure, while the whole of the right occipital lobe was converted into a colloid, extremely vascular mass, giving way under examination, this also extending anteriorly to the tumor as far as the fissure of Sylvius. The temperatures at the different stations were as follows:

Frontal, left	95° 75′.
Frontal, right	98° 33′.
Parietal, left	95°.
Parietal, right	99° 75′.
Occipital	96° 75′.
Occipital, right,	100° 60′.

The average of the left side was 96° 16′, and of the right was 99° 52′, and of the whole head 97° 84′. Since then Mills has reported two cases, one a case of frontal tumor, another a gumma involving the basal termination of the corpus callosum, its peduncles, the lamina cinerea, and the anterior perforated spaces, and Eskridge a case of tumor of the cerebellum with monocular hemianopsia. Mary Putnam Jacobi has also reported a case of tubercular meningitis. In all of these my statements were fully confirmed. Maragliano and Seppilli, of Reggio-Emilia, Italy, have made a series of investigations upon the cerebral temperature of the insane and the sane. They ascertained that it was exceptional to find the average temperature of the head higher than normal in simple melancholia and dementia; that the highest temperature is found in furious mania, then in decreasing ratio in melancholia agitata, paralytic dementia, agitated dementia, imbecility and idiocy, simple mania, simple melancholia, simple dementia; that in all mental diseases the occipital lobes are of lower temperature than the others; the temperature of the frontal lobes, which equals that of the parietal in agitated dementia, idiocy, and imbecility, is higher in mania, in simple melancholia and simple dementia, whereas in paralytic dementia and agitated melancholia the temperature of the parietal lobes is higher than that of the frontal. In all the principal groups of mental disease the averages of the two side of the head are almost equal, with the exception of congenital forms, in which the right shows a higher figure than the left. The general temperature of the body of the insane taken in the axilla and rectum is higher in melancholia agitata and furious mania, and generally goes on decreasing in regular order in paralytic dementia, agitated dementia, simple mania, idiocy and imbecility, tranquil dementia, simple melancholia. These Italian authors found the cerebral temperature of the sane to be higher than I did, their figures for the individual lobes being on the average $1\frac{96}{100}°$ greater than mine, whilst they found the whole head $1\frac{92}{100}°$ warmer. They explained this disparity by the fact that they did their work in June, July, and August, whilst mine was done in the winter and early spring, so that when they made some observations in December their figures were very nearly those that I had given, and they thus themselves admit that the disparity is explained by the difference of the tem-

perature of the surrounding atmosphere. Maragliano ascertained that thermometers placed on the skull were accurate indices of the temperature of the contents of the skull. He filled a skull with water at different temperatures, leaving the integument and hair in place, and found that the thermometers placed externally followed faithfully the oscillations of the temperature of the water within as denoted by thermometers placed therein. Amidon has made some interesting observations confirming my results. During the seventeen years that have since elapsed I have made a number of observations, and have had no reason to modify my original conclusions. I have not, however, found that cerebral thermometry is always of value, inasmuch as there are many organic diseases of the cerebrum in which the alteration in surface-temperature is not sufficient to lead to a positive diagnosis. In all cases, however, in which I found a variation to the degree which I have indicated I always found organic disease; so that in doubtful cases of intracranial neoplasm cerebral thermometry may be of great value when there is a local rise of temperature implicating one or more stations on one side of the head. Absence of rise, however, is of no diagnostic importance one way or the other.

CHAPTER III.

DISEASES OF THE PERIPHERAL NERVES.

NEURALGIA.

Definition. Neuralgia is a functional affection of the sensory fibres of the peripheral nerves, and manifests itself by pain.

Clinical History. The pain of neuralgia varies extremely in degree from a mild ache to a severe pain, and also in quality, running through all the gamut of sensations of which the sensory nerves are capable, so that it may be boring, dragging, burning, stabbing, shock-like, creeping, etc. The pain of neuralgia has certain characteristics that distinguish it from pain in a nerve due to organic cerebral, spinal, peripheral, or visceral disease, and these peculiarities are the tendency to shift from one nerve or set of nerves to another, and the presence of certain tender points (*points douloureux*). The tendency to shift manifests itself in the most remarkable manner at times, whilst the tender points are almost invariably found at the levels where the nerves pass from a deeper to a superficial level. Every nerve in the body may be the seat of neuralgia, so that a catalogue of all the different forms would be an anatomical list, but for practical purposes neuralgias may be divided into those of the head, trunk, and neck, the upper extremities, the lower extremities, and the visceral neuralgias.

NEURALGIA OF THE HEAD.

The neuralgias of the head are migraine and trigeminal neuralgia. Migraine will be considered in a special chapter, because it is more of a neurosis than a neuralgia. The trigeminal neuralgias are the ordinary neuralgia of the trigeminal nerve and *tic douloureux*. The ordinary trigeminal neuralgia may manifest itself in either of the three branches of the fifth pair, or in all the three branches simultaneously. The painful points in the ophthalmic division are (1) the supraorbital, at or a little above the supraorbital foramen; (2) the palpebral, in the upper eyelid; (3) the nasal, at the junction of the nasal bone with the cartilage; (4) the ocular, vaguely in the globe of the eye; (5) the trochlear, at the inner side of the orbit. In the second division of the fifth, the supramaxillary branch, the painful points are the cheek, eyelid, the side of the nose, the upper lip, zygoma, upper temporal region, the upper row of the teeth, and in the nasal cavities and gums; whilst in the infraorbital branch of the second division the painful points are (1) the infraorbital; (2)

malar; (3) superior labial; (4) the palatine. When the third division or inframaxillary division of the fifth is affected, the painful points are (1) the temporal; (2) the inferior dental, and, rarely, the inferior labial and to one side of the tongue, the so-called lingual point.

In all these simple forms of neuralgia, while the pain may vary in degree, as I have said, from a mild ache, coming on at certain irregular times, to a severe and shock-like pain, yet it should be sharply distinguished from that variety to which has been given the name of *tic douloureux*, in which the pains come on in paroxysms of great severity, so that the patient holds the face and sits in mute agony, and which, being very intractable and apt to continue through years, is entirely different in course and prognosis from the simpler forms.

NEURALGIAS OF THE TRUNK AND NECK.

The neuralgias of the trunk and neck are—

Cervico-occipital;
Phrenic;
Cervico-brachial;
Dorso-intercostal;
Lumbar.

Cervico-occipital neuralgia is in the regions of distribution of the four upper cervical nerves, and is therefore felt in the occipital and posterior parietal regions to the vertex as far forward as the parotid, and down the neck to the clavicular and upper thoracic regions, or it may be limited to the area of the great occipital nerve, the small occipital nerve, the inferior subcutaneous nerve, and the supraclavicular nerves.

In phrenic neuralgia the pain is at the lower and forward region of the thorax, at the points of insertion of the diaphragm, and at the mid-portion of the neck, where the phrenic nerve branches off from the cervical plexus, as well as in the chest and in the shoulder.

Dorso-intercostal neuralgia is in the area of the sensory branches of the dorsal nerves, mostly in the anterior divisions, and the tender points are found laterally in front and behind.

Lumbar neuralgia has its site in the first four lumbar nerves which supply the loins, the inguinal regions, the hypogastrium, the mons veneris, part of the scrotum and the labia majora, the antero-lateral and median regions of the thigh, the anterior portion of the knee-joint, the median surface of the leg, and the inner border of the foot to the great toe. It is rare to find simultaneous implication of all of this region, and it has therefore been divided into two groups, lumbo-abdominal neuralgia, or lumbago, and femoral neuralgia, the former being by far the most frequent. There is a form of neuralgia due to urethral stricture, or disease of the prostate or seminal vesicles, which is very apt to be confounded with lumbago, and yet which I have never seen described. It follows the pelvic lesions mentioned, often in neurotic or impressionable individuals, and is very intractable and difficult to treat, as I shall state further on.

NEURALGIAS OF THE UPPER EXTREMITIES.

The neuralgias of the upper extremity belong to the cervico-brachial group and nerves in the branches of the brachial plexus or the posterior branches of the four lower cervical nerves, so that the pain is in the upper arm or forearm, often extending into the hands and fingers, and the painful points are (1) the axillary point corresponding to the brachial plexus; (2) a scapular point; (3) a shoulder-point at the emergence of the cutaneous branches of the circumflex through the deltoid; (4) the median cephalic point, at the bend of the elbow answering to the musculo-spiral nerve; (5) an external humeral point three inches above the elbow at the emergence of the cutaneous division of the musculo-spiral; (6) the superior ulnar-point upon the ulnar nerve between the olecranon and the epitrochlea; (7) the inferior ulnar point, upon the ulnar just in front of the annular ligament of the wrist; (8) a radial point, where the radial nerve issues at the lower and outer part of the forearm.

But neuritis of these nerves is much more common than neuralgia.

NEURALGIA OF THE LOWER EXTREMITIES.

Neuralgia of the lower extremities consists chiefly of sciatica or pain in the sciatic nerve, and the painful point, as Gibney has shown, is usually best found in the following manner: Placing the thumb upon the great trochanter of the femur, and the second finger upon the tuber ischii, press down midway between the two with the index-finger, when the sensation of the patient will be a guide to having reached the sciatic nerve.

VISCERAL NEURALGIAS.

Visceral neuralgia is mainly of the kind known as colic, or accompanying organic affections of the intestines, the liver, or the female pelvic organs.

In all the different forms of neuralgia the various functions of the different nerves, motor, sensory, and sympathetic, may be affected. Thus, in trigeminal neuralgia there may occur spasm of the facial muscles, pallor or redness and even swelling of the face, increased lachrymation, salivation, herpes, changes in the hair, periosteal alterations, even erysipelas and neuro-paralytic ophthalmia, whilst hyperæsthesia and anæsthesia may sometimes be temporarily present; and in all the neuralgias analogous symptoms to these are common.

Pathology. A great deal of energy has been spent in discussion as to whether neuralgia is due to changes in the central cells of the sensory nerves, in the nerve-fibres themselves, or in the end-organs, and also as to whether these changes are secondary to vaso-motor disturbances; but it is useless to go into these differences of opinion, because they are entirely speculative, with scarcely a respectable fact to prop any one of them up. The truth of the matter is, that we do not know the molecular changes which constitute neural-

gia, and we never shall know until we have instruments delicate enough to enable us to dip down into a living cell of cord or skin and have a microscopic view of molecular life, or until we have gained such knowledge of all the cells of peripheral nervous structures that we can appreciate a slight departure from the normal. The congestions and the shifting and turnings of the nerve which were described by the earlier authors were really cases of neuritis, and not neuralgia. But although we do not know the molecular changes which are at play in a pain-suffering nerve, we do know many of the factors which can set up that pain.

Causation. The causes of neuralgia may be:

Heredity;
Gout;
Neurasthenia;
Anæmia;
Malaria;
Eye-strain;
Dental disease;
Disease of antrum;
Genital irritation;
Neural tumors;
Trauma;
Influenza;
Changes of temperature;
Toxic influences;
Kidney disease;
Syphilis;
Hysteria.

Heredity is often a cause of neuralgia, and, indeed, most of the cases of generalized or recurrent neuralgia will be found to be hereditary, although it must be confessed that this hereditary causation may well be overestimated. I do not believe, for instance, that a person has ever lived who has not had neuralgia at some time, and yet it would not be fair to say that this individual could transmit neuralgia hereditarily, as could a person who was constantly subject to the malady. This wide distinction between the constantly neuralgic individual and the occasionally neuralgic one is generally lost sight of by patients in giving a history of their heredity. But aside from a purely neuralgic heredity, neuralgic patients will very frequently have a neurotic heredity, such as epilepsy, migraine, neurasthenia, insanity, etc.

Gout is a frequent cause of neuralgia. But the gout which is described by English authors is seldom met with in this country, even among people living luxuriously and daintily, and it is more apt to be relatively slight joint-aches and disturbance of the chylopoïetic system than the full-fledged attacks of the disorder which the text-books describe. In many neuralgic individuals, however, whose parents have not been gouty, there has been a tradition of gout in the family

The neurasthenic is subject to aches and pains that are generally

vaguely neuralgic, but at times he has outspoken attacks of neuralgia.

Syphilis is not so frequently the cause of neuralgia as the abundant literature upon the subject would seem to prove, but, of course, it undoubtedly occurs. The intracranial exacerbations of this disease upon which so much diagnostic stress is laid are, however, so common in many forms of neuralgia as to have but little weight in my estimation.

Anæmia should always be sought for in any case of neuralgia where the cause is not certain, and reliance should not be placed upon the color of the lips, the face, and the conjunctiva, but examination should always be made of the heart and the great vessels for the characteristic blowing-sounds, and in doubtful cases an examination of the blood should be made.

Malarial neuralgia occurs in two forms: in the first place, with a regular periodicity, and, in the second place, as a sequel of malaria; and a sharp distinction should be made between these two forms for purposes of treatment. The periodicity of malarial neuralgia will sometimes have to be carefully looked for. Thus, I have known an obstinately recurring neuralgia to resolve itself under patient examination into an attack every second day at 9 o'clock in the morning, to which was added on every fourth day an attack at 2 o'clock in the afternoon, and it was not until the antiperiodic medicine was carefully administered at the time immediately preceding these periodical occurrences that relief was obtained. Nor is it always the case that a rise in temperature will accompany the periodical returns, so that absolute reliance cannot be placed upon this means of diagnosis. It is frequently said that the brow-ache is characteristic of malaria, but this statement should be viewed with some caution. It is undoubtedly true that brow-ache is observed in malaria, but this should not blind us to the fact that neuralgia of other nerves of the body may also occur in the same disease, especially of the different branches of the fifth pair and the sciatic, so that too much dependence should not be placed upon the anatomical localization of the neuralgia as a diagnostic factor.

In any case of obstinate neuralgia about the trigeminal nerve a careful examination should be made for errors of refraction and muscular insufficiencies of the eye-muscles; but if I may judge by my own experience, these are rare causes. In any case, however, it is well to remove any possible reflex irritation from the eye.

Dental disease is a frequent cause of neuralgia of the different branches of the trigeminus, and in any case of pain commencing in the jaw and radiating through the cheek a careful examination of the teeth should be made by a competent dentist. Any doubtful filling should be removed and repacked. Any persistently painful teeth or any from which the pain starts should be drilled into, even if outwardly and above they seem perfectly sound, for I have in several instances known a tooth which had been pronounced healthy yet to have imbedded in its fangs a mass of pus and gas that was incredible in its fetidness when provided with an outlet, and

these cases had had recurring neuralgias for several years. On the other hand, the mere presence of pain generally through the teeth, even though the teeth may have commenced to decay, is no reason whatever why they should be removed in the wholesale manner that is so often done. Nor will such removal usually give even temporary relief.

Disease of the antrum occasionally causes neuralgia of the trigeminus, and it should, therefore, not be overlooked in any case of persistent pain in the fifth pair.

The subject of genital irritation has been much exploited in the last few years as the cause of all sorts of nervous disorders, among others of neuralgia, but in my experience it is one of the minor causes. Of course, any well-marked vaginal, uterine, or ovarian disease may give rise to neuralgia, but the case then is not one of neuralgia, but of the malady which sets up the pain, whilst slight disorders of the genital organs seldom cause neuralgia unless joined with causes of general debility.

When neuromata cause neuralgia, the pain is generally limited to the nerve-trunk or branch upon which the tumors sit. In all cases of neuralgia, however, it is wise to make careful search for a possible tumor, which can usually be found with ease about the scalp, neck, rectum, female pelvis and trunk, and upper and lower extremities.

Trauma setting up neuralgia usually does it by implicating a nerve in a cicatrix, or by fracturing a bone and imprisoning the nerve in it or in the callus which is occasioned. I have never seen trauma induce neuralgia by itself without some local lesion of this kind, although neuralgia is a constant accompaniment of traumatic neurasthenia.

The late epidemic of influenza which has swept around the world has been a cause of neuralgia, mainly in this country of the dorso-intercostal form, and I think that this form should be considered as one of the diagnostic points of influenza.

Changes of temperature from heat to cold, sometimes from cold to heat, and barometric disturbances, are frequent causes of neuralgia, as are the changes of temperature and environment constituting the main factors in the process of acclimation, which, in a minor degree, is seen by us New Yorkers every summer on the arrival of our city-dwellers at the mountain and seashore, and again in the autumn upon the return of these individuals to the city. Some persons are most affected by the approach of a storm, others by the passage of it, whilst some of the most painful forms of neuralgia—those of the character which the French call *foudroyant*—are seen after a storm, during the presence of what is called the anti-cyclone, which is a period of blue skies, bright sunshine, high barometer, and slight atmospheric motion. It is well to study in any intractable case the behavior of the patient to these barometric disturbances, so as to be on the watch for severe attacks and know about when to anticipate them.

Various toxic influences may cause neuralgia, such as arsenic, lead,

ordinary illuminating-gas, some sewer-gases, alcoholism, and diabetes. In any obstinate case careful examination should be made of the urine, both quantitatively and qualitatively, and microscopically, if necessary, for I have had a number of cases of neuralgia come to me in individuals suffering from fatal nephritis, which had not been diagnosed. Usually, however, the pallor, the œdema of the eyelids and lower extremities will cause a suspicion of the real trouble, but in the cases of which I have spoken there were none of these outward signs, and the high arterial tension, to which attention should always be paid, was one of the first symptoms to arouse my suspicion.

Hysteria is a frequent cause of neuralgia, so that it should not be forgotten that genuine pain may exist in an hysteric.

Diagnosis. The study of the causes which I have just dwelt upon will make apparent many points of diagnosis, which I need only allude to again; thus, it should always be borne in mind that a so-called neuralgia may really be anæmia, neurasthenia, malaria, eye-strain, dental disease, disease of the antrum, genital irritation, or neuromata. In other words, neuralgia is but a symptom, and in all cases we should ask what it is a symptom of. The different causes which I have enumerated should be carefully thought of, and the diagnosis made thus by a discovery of some one cause or by exclusion of many. The diseases which are most apt to be mistaken for neuralgia, however, are—

Brain-tumor;
Intracranial syphilis;
Spinal tumors;
Pott's disease;
Spinal caries;
Neuritis;
Topoalgia.

A brain-tumor may cause either a general headache or pain limited to one distribution of a nerve, and be so slight as to picture well a neuralgia. Thus, in one patient who died in the course of six weeks with a tumor of the centrum ovale as big as a medium-sized potato, the symptoms at the onset were entirely those of a slight neuralgic pain over the forehead; whilst in another case of cerebral tumor, the patient dying in six months with a melano-sarcoma as big as a hen's egg imbedded in the temporo-sphenoidal lobe, the only symptom for the first four weeks had been that of neuralgia limited to the supraorbital branch of the fifth pair on the same side as the tumor; and in still another case, of a patient with a tumor of the cerebellum, the first symptom had been recurring attacks of obstinate neuralgia of the vertex. The diagnosis, however, can usually be made by a careful study of the case. A neuralgia should always excite suspicion when it appears gradually or suddenly in a patient who has not been subject to it, with some slight motor or sensory symptoms of other nerves; with some mental alteration perhaps; with attacks of nausea or vomiting; or subsequently to the appearance of tumors in other parts of the body. If an optic neuritis occurs, or any slight changes in the optic disk, the diagnosis is still more easily made; but

unfortunately in many cases of tumor optic neuritis is either a late symptom or does not appear at all. The development of a case of brain tumor, with its pronounced motor and sensory paralysis and mental symptoms, will inevitably make clear a diagnosis that may have been obscure at the start.

Intracranial syphilis in many of its forms is very frequently mistaken for simple neuralgia, but the diagnosis can be made in many instances by the three symptoms which I have discovered, and of which I speak at length in the chapter upon that subject, namely, the quasi-periodical headache, either nocturnal or at some stated time of the day, and the obstinate insomnia; the headache and the insomnia disappearing upon the supervention of any convulsive or paralytic symptoms. But it is in the early period of the headache, when it is only accompanied by obstinate insomnia, and before the supervention of convulsions or paralysis, that the diagnosis must be made. Great suspicion, therefore, should attach to any violent headache accompanied by obstinate insomnia, and careful search should be made for large glands of the head, neck, and groins, and the patient should be carefully interrogated as to any syphilitic infection; but even if no suspicious glands or history of infection are found, the patient should be put upon proper syphilitic treatment. In any case of obstinate headache not yielding to ordinary analgesics, whether a specific history of syphilis is given or not, syphilis should be regarded as one of the possibilities.

The pain of spinal tumors or caries is a fixed, severe, and persistent pain, limited to the distribution of one or two nerves, and is accompanied by local tenderness of the vertebra and by vertebral immobility, to which in time may be superadded symptoms of anæsthesia and paralysis.

The differentiation of Pott's disease from neuralgia is a matter of no difficulty when the former malady is well developed, and even in its early stages careful examination will detect some vertebral immobility, deviation, or tenderness, or some impairment of motion or sensation beyond the possibility of a neuralgia, or some muscular atrophy, or some marked alteration in the reflexes.

The diagnosis between neuritis and neuralgia is usually an easy one, but it may sometimes become a matter which requires careful consideration. In neuralgia the pain is apt to leap from one nerve to another, or even from one side of the body to the other. It is seldom accompanied by anæsthesia, and never by muscular atrophy, motor paralysis, glossy skin, or reaction of degeneration, whilst all of these latter symptoms are usually present in neuritis.

Topoalgia is the name given by Blocq and Onanoff to pain in different regions that are not anatomically or physiologically delimited. It may occur by itself, but is generally accompanied by neurasthenia, hysteria, or hypochondria.

PROGNOSIS. The prognosis will depend upon the kind of neuralgia and upon the cause; but as a general thing the prognosis is excellent, excepting where the cause is not removable either by medication or the surgeon's knife. Neuralgia of the trigeminus is of good

prognosis, except when it occurs in the form of *tic doulourcux* or accompanying great facial spasm. Other neuralgias of the head have also an excellent prognosis. The prognosis of the neuralgias of the upper extremities is almost invariably excellent, too, as is the case also with neuralgias of the trunk, excepting in that curious form which seems to bear some relation to antecedent urethral, prostatic, or seminal lesions of the male, which may be mistaken for ordinary lumbar neuralgia, and of which I have never seen a case cured. The neuralgias of the lower extremity are usually of doubtful prognosis, especially of the sciatic nerve; mainly, I think, because of the fact that it is so extremely difficult to put the limb at rest, for when this is done they improve as quickly as do neuralgias in other parts of the body. Visceral neuralgias are, as a rule, of good prognosis if they are not connected with some irremediable visceral lesion.

Treatment. Before entering upon the treatment of a case it may be well to realize that for therapeutic purposes all forms of neuralgia may be divided into two great classes; namely, those depending upon general causes weakening the whole organism, and those dependent upon local irritations; whilst occasionally a neuralgia may be produced by both causes, the general condition acting as a predisposing, whilst the local irritant acts as the exciting cause. I think that a great many difficulties may be cleared away when these statements are thoroughly understood. In every case, therefore, our first duty should be to search for the local irritant—such as a neuroma, a cicatrix, a fracture, a callus, a tumor, a marked error of refraction, or great muscular insufficiency of the ocular muscles—and remove it if that be possible, and then, if the neuralgia persists, take measures to bring the vitality of the whole organism up to the plane of health. To effect this latter purpose is often a work of art. If anæmia is present, it should be removed. Anæmia sufficient to cause persistent neuralgia must usually be treated with vigor, and the best plan, in my opinion, is the following: Cut off the expenditure of energy in the patient by one-half, either by keeping him in bed until noonday and having him go to bed early in the evening, or by forbidding all exercise whatsoever, ordering the use of a carriage in going around; or by making him rest three or four hours every afternoon. The morning rest is usually the best, however, inasmuch as anæmic patients are generally at their worst in the morning. Use large doses of iron, either the dialyzed iron, ʒj after meals in a cup of water; Dree's albuminate of iron, ʒj also after meals in a cup of water; or the peptonate of iron in the form of compressed tablets, gr. ij–iij after meals, three times daily; or the subcarbonate of iron, gr. xx–ʒj, when a tasteless powder is desired for children, to put in the food perhaps, or when a cheap preparation is needed. Lately I have been using ferratin, gr. iv–viij three times daily, in capsule, because it not only has no constipating effect, but seems to be slightly laxative at times. Tonic doses of quinine should be given with the iron, or five grains of sulphate of quinine every night at bedtime. Administer large

quantities of red meat, having it prepared hot at the three meals of the day, and in addition use a strong beef-tea, making a pound of meat into a pint of beef-tea, dividing it into three portions daily; if needed, also use use bullock's blood, obtained fresh from the abattoir every morning, and frozen hard into cakes and thus eaten, which, gruesome as it may sound, is by no means unpleasant to many people. Administer small doses of bichloride of mercury, $\frac{1}{64}$ to $\frac{1}{32}$ of a grain three times a day. If there should be malaria, this must be carefully removed. If the neuralgia come in the malarial paroxysms, the treatment is quite different from what it is when the neuralgia succeeds this. The malarial paroxysms must be broken up by large doses of quinine (ten, twenty, or thirty grains, according to how the patient is known to bear the drug or the severity of the malaria), administered a couple of hours before the attack is expected; or if the attack occurs very early in the morning before waking, the quinine should be given at bedtime, and the dose should always be sufficient to produce distinct cinchonism. When the paroxysms have disappeared, the quinine may be gradually reduced, and when the dose has become very small it may be left off entirely, and arsenic given for a week or two—best Fowler's solution of arsenic, three drops three times a day, in a wineglass of water, or arsenious acid, $\frac{1}{100}$ grain three times a day. Some individuals, however, can take neither quinine nor arsenic, although in my own experience those who cannot take quinine in truly malarial paroxysms are very few in number. In such a case cinchona bark may sometimes be borne, preferably in the form of the elegant elixirs which are prepared by the leading pharmacists of our large cities, the dose varying from ℥ss to ℥iv, according to the amount of cinchona used; or the tinct. cinch. comp. (*U. S.* and *B. P.*), is a cheap and effective preparation for the lower classes, but it is very disagreeable to people of delicate palates. Its dose is ℥j three times daily. If cinchona will not answer, the iodide of potash in continuous doses of ten to fifteen grains three times a day, or the tincture of iodine fifteen to twenty drops at a dose, three times a day, in a tumbler of water, or lemon-juice, ʒj–℥iij in a glass of water, steadily used two or three times a day, may one of them answer the purpose of overcoming the periodicity; or arsenic may do in conjunction with cinchonidia or chinoidin, and I have found the following prescription of considerable value:

℞.—Chinoidin . . . . ʒij.
Hydrastiæ . . . . ʒj.
Ferri sulph. exsic. . . . . . . ʒj.—M.

Ft. in pil. no. xxx. S.—One three times daily one hour after meals.

If arsenic cannot be taken after the quinine in antiperiodic doses has been stopped, resort may be had to the foregoing prescription, or the patient may be put upon dilute nitro-muriatic acid, 20 drops three times a day, after meals, in a wineglass of water; or an alkali may be used, even so mild a one as Vichy or Giesshuebler; or a change of climate may be tried. In the neuralgia which may have succeeded

malaria, quinine may be of great value, or it may be perfectly useless, and a test must be made to determine this point. In many of these cases the pain is kept up by anæmia, which can be readily removed by one of the preparations of iron just mentioned. If this should prove useless, the acids or the alkalies will be all the treatment that is needed, although a change of climate is usually beneficial, provided that the climate selected is one that usually agrees with the patient. The treatment of gout or lithæmia (which is the American gout) should be in accordance with the rules laid down in speaking of lithæmic neurasthenia (Chap. IX., "Neurasthenia"), and to the same chapter the reader must turn for directions as to the treatment of the neuralgia of neurasthenics. I have much more faith in the general treatment of neuralgia than I have in the local treatment of it; in other words, I believe that neuralgia is almost invariably the cry of a debilitated organism—not of impoverished blood, as was said many years ago at one stage of our knowledge—oftener than it is the cry of an independently damaged nerve. In every instance, therefore, the general treatment should be given the preference. If, however, it be impossible to carry this out, or if the pain is persistent in spite of the general treatment, then local measures for the relief of pain should be used—very cautiously, however, and not with the free hand with which they must be employed in neuritis, as I shall have occasion to point out. The best analgesics, in my estimation, are: opium and morphine, codeia, phenacetine, antipyrin, aconitia, aconite, aquapuncture and acupuncture, veratrum viride, counter-irritation by blisters and rubefacients, hot applications, and electricity. Of all these, by all means the best is opium in the form of its distinguished extractive, morphine, and the best of all the morphines, in my opinion, is the sulphate, although it may be that some people in whom the sulphate cannot be employed will bear the muriate well, but it is very seldom that the sulphate cannot be borne if care is taken, as it always should be, that the opium is obtained from some perfectly reliable manufactory in packages marked with the guaranteed analysis. The opium and morphine that are usually found at the druggists are very unreliable drugs, and not only do I take particular care that all used by my patients should be obtained as I have directed, but also that the solution of it shall be made fresh every few days, put into bottles with glass or cork stoppers, or the tablet triturates shall be put into a bottle with a screw top, and that over them, as they lie in the bottle, shall be placed a thick pledget of cotton. The hypodermic administration of morphine is by far the best means of relieving acute pain. Unfortunately, however, it is a very dangerous drug to use with neuralgics, and I make it a rule never to employ it except in such acute pain that the paroxysms cannot otherwise be controlled, and then I never let the patient use the hypodermic himself. In some cases morphine will have its effect enhanced by the addition to each dose of $\frac{1}{100}$ grain of atropine, as will be pointed out shortly, whilst in other cases the effect is diminished by this combination. Codeia is, next to morphine, the most valuable extract of opium for its effect upon the visceral neuralgias. Phenacetine has not been a reliable drug in my

hands except in dorso-intercostal neuralgia, especially when caused by influenza. Antipyrin is undoubtedly a most effective analgesic, but it is also a depressant that is dangerous in most cases of neuralgia, except for temporary use; indeed, I have seen so many cases of neuralgia seriously depressed by the use of antipyrin that I seldom administer it. Aconitia is sometimes an excellent analgesic, more especially in trigeminal and sciatic neuralgia, and Duquesnel's and Merck's preparations are the best. These can be obtained from all the large manufacturing houses in the form of hypodermic tablet triturates. The dose should be $\frac{1}{150}$ grain to start with, given every four or five hours, and gradually increased until some slight tingling or numbness is perceived by the patient, and for this purpose it is safe to give as high as $\frac{1}{50}$ or $\frac{1}{25}$ grain, but it should never be administered to a person with arterial, cardiac, renal, or pulmonary disease. It may be continued in suitable individuals for weeks, and to show how well it is borne I may cite the case of a patient who was taking $\frac{1}{20}$ grain four times a day for *tic douloureux*, when he was caught in the terrible crush upon the Brooklyn Bridge that occurred shortly after the opening of the structure, and which resulted in the loss of several lives; and yet his heart was not affected in any appreciable degree by the terrible scene. Aconite is an excellent analgesic as a local application, either alone or in combination with other substances, as in the following prescription:

℞.—Tr. aconiti rad.
Tr. opii
Tr. iodi } . . . . . . āā ℥j. M.

S.—Apply with camel's-hair brush.

But it should not be used internally for this purpose, because of its depressing effect. This is also true of veratrum viride. Acupuncture and aquapuncture are occasionally excellent analgesics where the pain is localized, and this not only in hysterical individuals, for I have seen it relieve pain marvellously in certain cases that were in no sense of the word hysterical or even unusually sensitive; and patients who crave a hypodermic will sometimes be satisfied with acupuncture or aquapuncture. Warmth by means of hot or dry applications is not so much a reliever of pain as an aid to the true relievers of pain. Neuralgic nerves, as a rule, do best in an equable and warm or moderate temperature. The best means of applying warmth is by poultices, encasing the body or limb in oil silk or wool gauze, or by the application to the neuralgic part of a handkerchief dipped into boiling hot water, quickly twisted perfectly dry by a stick in each corner, and removed in a few minutes as the heat becomes bearable. Electricity is of great value in some forms of neuralgia, and perfectly useless in others, as will be pointed out shortly.

We will now proceed to detail the application of these general and special considerations to each form of neuralgia.

In ordinary trigeminal neuralgia the general health must be carefully attended to, and all causes of local irritation should be removed, but usually, even after this has been done, some neuralgia will persist.

This can be relieved by aconitia, quinine, either alone or combined with codeia (the former in tonic doses, the latter $\frac{1}{3}$ or $\frac{1}{4}$ grain, each three times a day), and arsenic, either Fowler's solution, 2 or 3 drops three times daily, or arsenious acid, $\frac{1}{100}$ grain three times a day. This has been an effective combination in my practice:

℞.—Quin. sulph. . . . . ℨj.
Acid. arseniosi . . . . gr. $\frac{1}{3}$.
Codeiæ . . . . gr. vij. M

Ft. pil. no. xxx. S.—One three times daily, one hour after meals.

The addition of gr. j–ij of salicylate of soda to each dose of this will increase the analgesic effect without making it more depressing.

Electricity in the form of galvanism or faradism, generally the former, is sometimes of use, but only exceptionally so. When galvanism is used, the neck electrode (Fig. 67) should be placed on the nape of the neck, and the other electrode of Fig. 71 should be placed upon the affected nerve-trunk, covering one or more of the tender points, and a current of 2 to 5 milliampères should be carefully measured off and passed during a sitting of three to five minutes. If faradism is used, the electrodes of Fig. 68 should be placed about two inches apart, and a gentle current, sufficiently strong to be pleasantly felt by the patient, should be administered from ten minutes to half an hour at a time, or the patient may take one electrode in the hands, whilst the physician takes the other in one of his and then applies his fingers gently to the patient's face, using sufficient current to be gently felt, and to cause a distinct crackling sound and sensation. This faradic application may be made for some length of time, generally fifteen minutes, but even thirty minutes to an hour in the severe cases. These electrical applications should be used every second day at least for a period varying from four to twelve weeks, according to the severity of the neuralgia and the relief that they may give. The electric air coming from the static machine is generally very refreshing to the patient, but it is a feeble agent for relieving pain or modifying the nutrition of the nerve, whilst the electric sparks are either inert or aggravate the suffering.

In occipital neuralgias of the head, however, the treatment is, curiously enough, quite different, for in them aconitia is unreliable, arsenic is valuable, quinine may be valuable or may aggravate, whilst electricity, especially galvanism, is usually of great value. The galvanism should be applied by means of the neck electrode (Fig. 67) placed upon the nape of the neck, whilst electrode Fig. 68 is placed upon the forehead, and a current gently turned on of 2 to 5 milliampères, always commencing with the lowest figure for the first sitting, and allowed to pass for from three to five minutes; whilst faradism may be applied as when the trigeminus is affected.

Neuralgias of the trunk, which are mainly of the dorso-intercostal type, are best treated by means of phenacetine, galvanization of the spinal cord, static current in the form of sparks, local support by means of a strong adhesive plaster, liniments, and in severe cases by

rest of the trunk in bed or on a lounge, the former best. Quinine in tonic doses (gr. ij–iij three times daily) usually has an excellent effect upon these dorso-intercostal neuralgias, especially when it is combined with moderate doses (gr. ij–iij) of a reliable salicylate of soda.

Neuralgias of the upper extremity are generally best treated by means of aconitia, quinine in combination with codeia or salicylate of soda, galvanization of the main nerve-trunks, and rest of the arm in a sling, together with such general treatment as may be indicated.

Neuralgias of the lower extremity, mainly sciatica, are difficult to treat satisfactorily unless the affected nerve is put completely at rest by confining the patient to a couch, stopping his walking, and in all possible ways restricting the use of the muscles of the lower limb, and this rest must be kept up for a period of from four to twelve weeks, according to the chronicity of the case. To this treatment should be added salicylate of soda, arsenic, galvanism, and the static current. Injections hypodermically of osmic acid are occasionally very beneficial, one to three drops of a one per cent. solution being used, and so are aquapuncture and acupuncture, but these remedies can never be relied upon. Another very important thing in all cases of sciatica is to see that the patient has a shoe that fits the foot properly, with a sensible low heel, a straight inner sole, and wide enough at the toes to permit the body being lifted on the toes, so that they will be spread without bulging over the edge of the sole, and will give all the wide base of support and all the play of muscle in foot, toe, and leg that Nature intended. This apparently small matter I have found to be of very great importance in a number of continuing cases of sciatica.

Visceral neuralgias are best treated in the acute paroxysms by absolute rest and a sufficient hypodermic of morphine, with or without atropine, according as is found best with the patient, and then afterward subnitrate of bismuth, salol, and codeia, or oxalate of cerium and codeia, as in the following prescriptions:

℞.—Bismuth. subnit.	ʒv.	
Codeiæ	gr. vj.	
Salol	ʒj.	M.

Ft. in chart. no. xxx. S.—One powder in a wineglass of water, three times daily.

℞.—Cerii oxalat.	ʒj.	
Codeiæ	gr. vj.	
Salol	ʒj.	M.

Ft. in pil. no. xxx. S.—One three times daily.

To these should be added warmth, hot or dry. In ovarian or uterine neuralgias, gelsemium, 5 to 10 drops of the fluid extract, will be found of great value.

In obstinate cases of neuralgias that have resisted all medicinal treatment, or in that affection of the fifth pair which is known as *tic douloureux*, or when there is any pressure upon a nerve by a neuroma, hypertrophied bone, or callus, it is often advisable to resort to surgical procedures of relief. The removal of a neuroma or of a

hypertrophied bone or callus is, of course, sufficiently simple and needs no discussion. When, however, the neuralgia cannot be traced to any gross and removable causes, such as these, nerve-stretching or nerve resection or section may occasionally give relief at least for the time being, and the patient may be willing to have the operation undertaken with the hope of relief that may possibly last only a few weeks, but that may possibly, on the other hand, last a year or two. Nerve-stretching is the least harmful of all these procedures, and there is no question that in a number of instances it has given most effectual relief for an indefinite period. I have known it to relieve sciatica in seven cases for a period of a year or more. But in the stretching of a mixed or motor nerve care should be taken not to stretch so much as to cause paralysis. The following table of Trambetta shows the amount of force necessary to rupture the different nerves:

Sciatic (trunk)	84 kilogrammes.
Internal popliteal (branch)	52 "
Crural	38 "
Median	38 "
Ulnar	27 "
Radial	27 "
Brachial plexus (in neck)	22 to 29 "
Brachial plexus (shoulder)	16 to 17 "
Infra-orbital	2½ "
Supra-orbital	2⅔ "
Mental	2½ "

Symington saw the sciatic ruptured in a robust subject by the weight of 65 kilogrammes, and in an emaciated girl who had died of phthisis with only 32 kilogrammes. No such force as is necessary to rupture a nerve should be employed, and it would be a safe thing to keep thoroughly well within the limit, because in three cases upon which I have operated myself I have seen paralysis of the facial result from a force that was entirely incapable of rupturing the nerve. In some cases, where there are spasmodic movements and where the nerve is unimportant, it is well to consider that a motor paralysis may be of therapeutic value in putting the muscles at rest, and I have recommended it in some cases with this end in view, and beneficially, as the results seemingly proved. Forcible flexion of the thigh upon the body has apparently at times acted as efficiently as nerve-stretching, especially in sciatica. The operative procedures that have been done for the neuralgia of the trigeminus which is known as *tic douloureux* are nerve-stretching, nerve-division, or neurotomy, the excision of a part of the nerve, or neurectomy, and nerve-avulsion. Sometimes the dental nerves are the source of infra-orbital neuralgia, and then a removal of the segment of the maxillary tuberosity containing the superior and posterior dental nerves has been recommended. Carnochan has suggested that in *tic douloureux* the trunk of the nerve should be resected beyond Meckel's ganglion, and that this ganglion should also be taken out. Horsley believes that it is better to remove the Gasserian ganglion, and has shown by dissection that this ganglion could be separated from the cavernous sinus. Horsley's case, in which he operated in this manner, however, died seven hours after the operation from shock. Horsley reports

nineteen cases of operations which he has done upon the different branches of the fifth pair, and in some cures were effected for three or four years, in others the pain disappeared for a variable length of time, in some there was no relief at all, and in one, which was the operation upon the Gasserian ganglion, there was death. My experience has been similar.

Chipault has collected 50 cases, with the following results:

Nine fatal results from operation. Seven from removal of Gasserian ganglion, 1 from partial removal, and 1 from intracranial resection of fifth.

Excision of Gasserian ganglion	25 cases.
Removal of semilunar ganglion	2 "
Resection of fifth and partial removal of Gasserian ganglion	6 "
Intracranial resection of fifth	16 "

Results: Excision of Gasserian ganglion.

Improved	7 cases.
Recovered	9 "
Failed	7 "

Results: Intracranial resection of fifth.

Improved	6 cases.
Recovered	4 "
Failed	4 "

Results: Resection and partial removal of Gasserian ganglion.

Improved	2 cases.
Recovered	2 "
Failed	2 "

The operative methods adopted in cases of removal of Gasserian ganglion were those of Rose, Navarro, Park, d'Artieda, Doyen, Horsley, Hartley, Krause, and Quenu. The methods of Horsley, Hartley, Krause, and Quenu are quite similar. In many of the cases reported as improved the improvement was only temporary or partial. Intracranial operation upon this nerve or the ganglion, as is evident, is an exceedingly dangerous and uncertain procedure.

These surgical procedures are, therefore, evidently a last resort, and should only be undertaken in such desperate cases as cannot be treated by medicinal means, and no positive promise should be held out to the patient as to what they will effect.

SIMPLE NEURITIS.

Definition. Neuritis is an affection of a nerve-trunk or nerve-branches causing, either singly or together, pain, impaired sensation, and motor paralysis and atrophy.

Causes. The causes of neuritis are—

- Traumatism;
- Cold;
- Certain diatheses;
- Implication of neighboring organs;
- Neuromata;
- Adenitis.

Wounds, contused or lacerated; dislocations, especially of the shoulder-joint; pressure, as by sleeping with the hand under the head;

railroad injuries—may all be traumatic causes of neuritis. Extreme contractions of muscles also exceptionally produce it.

Exposure to cold is a frequent cause of neuritis.

Gout, syphilis, cancer, leucocythæmia, smallpox, typhoid, diphtheria, and many other general diseases would seem at times to act as predisposing causes of neuritis, rendering them more liable to ordinary exciting causes.

Pneumonia, pleurisy, meningitis, articular rheumatism, and other joint-affections may cause neuritis by an extension of the molecular changes to the nerve-sheaths.

Neuromata are formations that are fully described in a separate chapter (Chap. "Neuromata").

I have seen a few cases in which adenitis, usually strumous in character, had seemingly been a cause of neuritis.

Clinical Symptoms. The clinical symptoms of neuritis are pain, impairment of the different sensations, motor paralysis, atrophy, and electrical changes.

These symptoms vary extremely in the different cases, even in the mixed nerves, whilst in the cranial nerves their variation depends, of course, upon the particular character of the nerve that is affected, neuritis of the optic nerve leading to changes of vision and optic neuritis; of the auditory, to deafness, loud noises, disturbances of equilibrium; of the third pair or motor oculi, to strabismus, disturbances of the pupillary muscles, etc. Neuritis of the intracranial nerves is seldom seen by itself, but usually in conjunction with the intracranial diseases, and occasionally also with multiple neuritis and atrophy from spinal lesions, so that in speaking of neuritis in this chapter I shall confine myself to neuritis of the mixed nerves. Neuritis of the sympathetic nerve-fibres is as yet a very obscure chapter in pathology, and affections which in the past have been supposed to be such are resolving themselves into ordinary neuritis in a surprisingly large number of cases. (*Vide* Chap. "Diseases of the Sympathetic Nervous System.")

Usually the first symptom is pain in the affected nerve. This will vary according to the degree of the neuritis. In slight cases the pain may be so slight as not to exceed that of an ordinary neuralgia, but in the majority it quickly becomes severe enough to cause great suffering, although there are remissions, so that it is bearable during the daytime and becomes severe at night, to such an extent as usually to interfere with sleep. There soon supervenes impairment of sensation and motility. The sense of touch and the sense of pain are usually most impaired, whilst temperature and the muscular sense are less frequently affected, although there are many variations from this rule. The tactile sense may be merely blunted, or it may be absolutely lost. The sense of pain in the slighter cases is usually but slightly affected, although the conduction of the painful sensation may be considerably retarded. The muscular sense and the sense of temperature vary extremely in the amount of impairment. Motor paralysis usually comes on quickly, and is succeeded by atrophy in the course of a few weeks. Trophic changes in the

skin are very constant, producing the so-called glossy skin, and when this is observed in the fingers they become tapering in shape, which alteration, together with the cutaneous glossiness, is almost pathognomonic. There may be increased perspiration, or this may be decreased. Fibrillary twitchings of the affected muscles are also occasionally observed. Herpes is not common in simple neuritis. Electrical alteration may be present both to the faradic and galvanic current, and present the phenomena which have already been described as the *reaction of degeneration* (Part I., Chap. II.).

Neuritis may be either acute, subacute, or chronic. Its duration varies exceedingly, and whilst it is in one sense a self-limited disease, its self-limitations do not follow any general law that is at present known. There is no disease that is more modified by proper treatment.

In the acute form there are sometimes chilliness or rigor, headache, insomnia, and slight febrile reaction, but the subacute and chronic cases have no general symptoms.

PATHOLOGY. Before entering into the pathological changes that constitute a neuritis we should consider briefly the structure of a mixed nerve. The axis-cylinder is a continuous structure that runs from the central to the peripheral organ. The motor nerve takes its origin from the anterior horn of the spinal cord or the motor nuclei of the medulla oblongata, and passes out to join with other filaments going to the end-plate that lies upon the muscular fibre. The ganglion-cells have already been spoken of (page 27), and it is in the nucleus or nucleoles of these that the motor nerve takes its origin. The termination in the muscles has been carefully investigated, especially by Gessler, in the green Italian lizard and the guinea-pig, and recently (1892) by Babes and Marinesco. Gessler

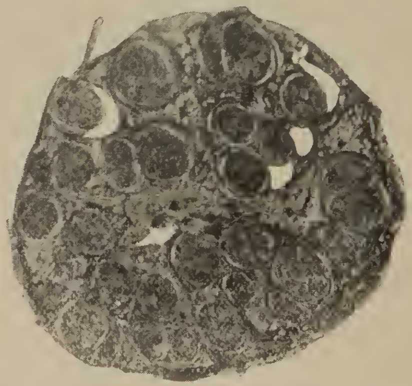
FIG. 105.

Normal sciatic nerve of frog. Obtained by teasing.

states that the motor end-plate is essentially the same in the mammalia and reptiles, and that it consists of a fine nerve-plexus, between or under which—for authors differ on this very materially—lies a finely granulated mass, containing two kinds of nuclei, the smaller ones being granulated and of irregular shape, the larger ones being oval with double contours and a nucleolus. The former are known as the *Scheiden-* or *Geästkerne*, or, as Ranvier calls them, the *noyaux*

vaginaux or *noyaux de l'arboration*, while the latter are the *Grund* or *Sohlenkerne*, or *noyaux fondamentaux* of Ranvier. To this motor end-plate, lying upon the muscle, come the medullated nerve-fibres by a right-angle, losing their medullary sheath as they pass into the nerve-plexus just described. The sensory nerve, on the other hand, takes its origin in the ganglion upon the posterior root, and passes down to the sensory structures of the periphery to Pacinian corpuscles, the tactile bodies, the end-bulbs, and the nerve networks (page 30). From its point of origin to its peripheral termination there is one structure of the nerve that runs continuously, the axis-cylinder, as has already been said. This is a highly nitrogenized body consisting of a number of primitive fibrils arranged longitudinally and glued together by a minutely granular substance. Around this axis-cylinder is a sheath of myelin, a fatty substance which does not pass with the nerve into either the central or peripheral end-organs, and which, moreover, is not continuous in its course, but consists of a series of tubes or segments whose points of junction are marked by a constriction in the contour. Finally, around the axis-cylinder and the sheaths of myelin is a connective tissue covering the sheath of Schwann, and this is also segmented, the point of constriction being known as the node of Ranvier. Occasionally, upon the axis-cylinder are seen certain lines which are known as arrow-markings, or markings of Schmitt. These were formerly thought to be arti-

FIG. 106.

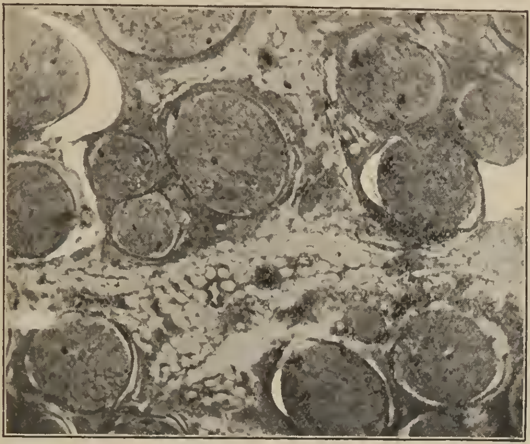


Section of the human median nerve, magnified 12 diameters.

ficial products, but recently it has been claimed that they are little masses of protoplasm, whilst still others maintain that they belong to the sheath of Schwann and are connective tissue. The sheath of Schwann has a few sparse nuclei. These different nerves are joined together by connective-tissue sheaths into bundles, forming a nerve-trunk or nerve-filaments. Freely anastomosing capillaries run within the nerve- and lymph-spaces, and are to be found within the nerve-sheath outside of the fibres. Connective-tissue cells and fine connective-tissue fibrils lie between the bundles of nerve-fibres. An excel-

lent idea may be gained by anybody of the changes that occur in neuritis by making a section of the sciatic of a frog, then excising pieces on different days after the original section, and observing the changes.

FIG. 107.

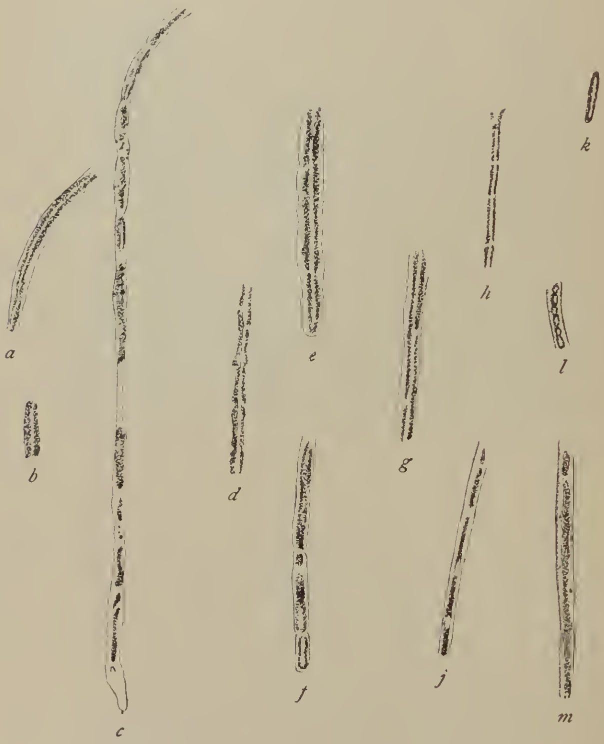


Section of the same nerve, magnified 22.5 diameters.

Fig. 108 is a sketch of such changes that were found on the fourth day after such section of a frog's sciatic. It will be seen that the axis-cylinder in some of these is entirely unaffected, and that the medullary sheath alone shows alteration, the sheath of Schwann in all being unaltered. The medullary sheath, as will be observed, manifests a tendency to break up into various irregular forms. In Fig. 109, which represents at A the same sciatic nerve on the eleventh day after section, these changes are seen to be much more marked, whilst the axis-cylinder in all is more or less affected. At later periods, as shown at B, representing the fifteenth day, these changes in the axis-cylinder and the medullary sheath increase in intensity, whilst the connective-tissue sheath of Schwann becomes somewhat opaque, and its nuclei, which are ordinarily sparse and slightly stained, so that it requires careful focussing to perceive them, multiply and imbibe more and more of the coloring-matter. These molecular alterations are similar to those which take place in a human nerve suffering from neuritis, if not identical with them. The changes in the myelin are supposed to be due either to a fatty or albuminoid degeneration or a process of saponification, and there is a diversity of opinion as to whether this latter is due to active migratory white blood-globules, and furthermore as to whether the white blood-globules may not be the connective-tissue cells. According to the degree of neuritis, the medullary sheath may be disintegrated in varying degree or may be entirely absorbed. It would seem quite probable that in all cases the axis-cylinder is affected in the human nerve, as the above figures show that it is in the frog's sciatic, although some

authors maintain that it remains intact, being simply deprived of its function. The numerous nuclei of the altered sheath of Schwann either come from a segmentation of the original nucleus of the segment, or are white blood-globules or proliferated connective-tissue cells.

FIG. 108.



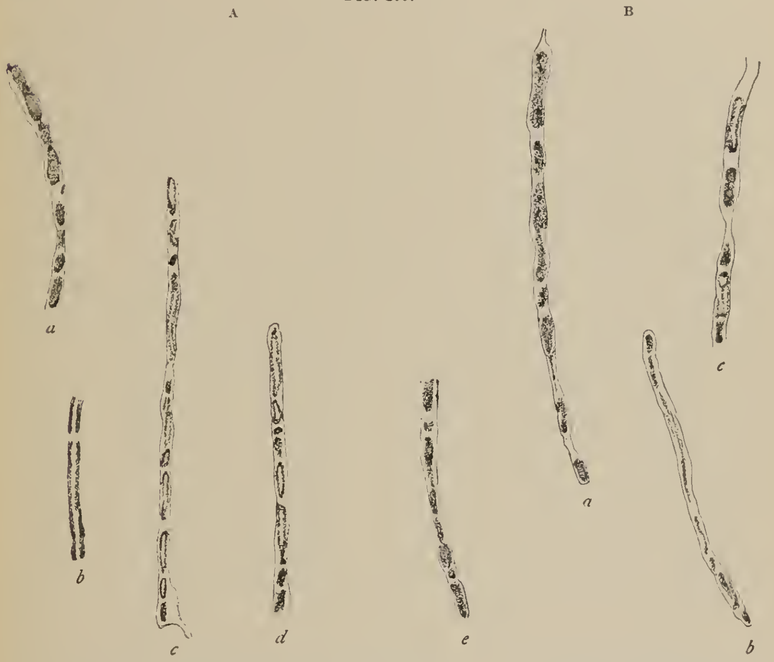
Changes on the fourth day after section of a frog's sciatic.

The inter-neural and peri-neural connective tissue may also become involved in the neuritic process. Considerable confusion exists as to what the methods of regeneration are in a nerve that has been afflicted with neuritis. By some it is held that the central segment is the part from which regeneration begins, whilst others maintain that this commences in the peripheral end, or that new axis-cylinders and medullary sheaths may develop from the nuclei in the sheath of Schwann. In some cases the neuritis affects the nerve-fibres, when it is known as parenchymatous neuritis; whilst in other cases the connective-tissue elements are mainly altered—so-called interstitial neuritis; and in still a third class of cases both the nerve-fibres and the interstitial tissue may be affected together.

PROGNOSIS. The prognosis of neuritis is generally good. A recent writer, Bowlby, states that it is invariably good, and that all cases recover; but with this I cannot agree, because it has been my expe-

rience to meet with a number of cases that did not recover, although I am inclined to believe that proper treatment will usually effect a cure if undertaken before the neuritis shows a tendency to become multiple and affect the other nerves. In traumatic neuritis, however, the prognosis may be modified by the nature of the trauma.

FIG. 109.



A. Changes on the eleventh day after section of a frog's sciatic.

B. Changes on the fifteenth day after section.

A clean cut across the nerve, for example, must be repaired surgically, or a nerve that is caught in a mass of callus or in an inflamed joint would have such pressure upon it as to make it impossible to treat it unless this were surgically removed. The duration is exceedingly variable.

TREATMENT. The primary necessity in the treatment of neuritis is to put the affected nerve at rest. I believe the reason that neuritis of the upper extremities and the trunk is invariably of better prognosis than neuritis of the lower extremities is due to the fact that it is so much easier to put the nerves of the upper extremity and the trunk at rest. Indeed, most cases of sciatic neuritis can be handled perfectly with proper rest, whilst without it the affection is usually very intractable. When there is a neuritis of the upper extremities the affected arm should be carried in a sling, the patient should have somebody dress and undress him, and even in some severe cases great benefit may be obtained by putting the patient to bed for a few weeks. In neuritis of the trunk every effort should

be made to lessen the movements of the trunk-muscles, and this can be obtained in a minor degree by having the patient abstain from walking as much as possible, or from horseback-riding, or from bending the trunk backward, forward, or laterally, or by putting on a strong strip of adhesive plaster, or finally by putting the patient to bed. Rest of the lower extremities can only be obtained adequately by putting the patient in bed, and this should always be done at the outset; and then, as the disease is brought under control, the patient should abstain as much as possible from walking or standing, and should, if his means permit it, go about in a carriage, or, if this is not possible, in street cars. In the early stage of a neuritis the pain is usually the most distressing element, and this can be overcome only by opium or some of its alkaloids, in conjunction with quinine. There is not the slightest use in wasting any time in this initial pain of neuritis upon such analgesics as phenacetine, antipyrin, or exalgine, or their congeners. Opium, and opium alone—and I say it boldly—can control this initial pain. We must, therefore, consider carefully the question as to whether our patient is of such a nature that he is likely to form the opium-habit, and if he is, we must look frankly in the face the question as to whether it is best for him to suffer the pain or run the risk of forming this opium-habit. It has been my experience that the patient is in no danger unless he is one of those weak individuals whom I have described elsewhere (Chap. "Alcoholism, Morphomania, etc."), who are prone to form habits to which they speedily become abjectly enslaved. In the many cases of neuritis that I have treated I have never yet made an opium-eater. Indeed, I think there is some subtle law at work here which prevents the person who is in real need of opium from becoming a slave to it. I always use the sulphate of morphine, taking care that it is prepared from a reliable opium, as most of the morphine of the shops is either from cheap preparations or has become stale. I use it hypodermically and commence with a dose of $\frac{1}{8}$ grain. Usually the pain of neuritis is worse at night or late in the afternoon, and this is the time when I give the hypodermic. If necessary, I increase the dose of morphine to the point that will relieve pain, and I have seldom had occasion to exceed $\frac{3}{4}$ grain at a dose. This will usually control the pain in a week or two, and then the morphine is gradually decreased in quantity. During this period the patient is instructed to take quinine, and the dose will vary according to the purpose I have in view. If the general health is poor, I give 2 grains of quinine three times a day as a tonic, perhaps adding 2 or 3 grains at the time of the hypodermic. If the general health, however, is good, I give 5 grains of quinine at the time that the hypodermic is given, and I prefer to administer it either in the form of capsules or fresh tablet triturates, which latter should always be tested by the physician by crumbling them between his fingers to see if they are fresh. Quinine in the form of solution with an acid is very prone to disagree with the stomach when long continued, and I have therefore almost abandoned its use. As the pain comes under

control, I withdraw the opium, as I have said, and then begin the use of the salicylate of soda in conjunction with the quinine, prescribing the former in doses of 2 or 3 grains three times a day. I seldom use larger doses of salicylate of soda, because I have not found that they add anything to the control of the disease, whilst they are very apt to depress the patient. If the quinine and the salicylate of soda are not sufficient to control the pain, I add to them moderate doses of phenacetine, 5 grains once or twice a day. It should always be carefully ascertained that the salicylate of soda has been carefully and reliably prepared, and this should be as carefully looked after as the quinine and the morphine have been. Exalgine in my hands has not been a drug of much value. Antipyrin undoubtedly relieves the pain better than either phenacetine, exalgine, or salicylate of soda, but the great objection to it is the depression, and I have for this reason almost given it up. Salophen will occasionally act very well in the gouty cases, in 5 grain doses two or three times daily. As the pain becomes a minor matter, electricity may be used with great advantage, but it should never be used before this period or it will be certain to increase the pain, often to an unbearable degree. A beginning should be made carefully with the galvanic current. I have given up attempting to localize the current to the affected nerve, because in order to do this we must make use of a very small electrode (Fig. 72), and this necessarily concentrates the electricity upon the nerve, and this concentration irritates it. I therefore prefer, when the neuritis is in the upper extremity, to bend electrode Fig. 68 over the shoulder, and bend electrode Fig. 69 around the wrist, and then very gently turn on a current of 2 to 3 milliampères, permit it to pass about three minutes, and carefully turn it off. Skeptics in the use of electricity need only try this at the proper stage in a few patients suffering from neuritis to satisfy themselves of the comfort and well-being that will be experienced for from twelve to fourteen hours afterward. In a neuritis of the lower extremities I also make use of electrodes Fig. 68 and Fig. 69, and place them so as to bring the whole limb into the circuit between them. If a nerve of the face is affected, or one hand or foot, the same principle should be made use of in the selection of suitable electrodes, taking care always to select as large ones as possible. The quantity of galvanism should not be increased for a week or two beyond 3 milliampères, but if the first two or three applications do not irritate the nerve, the sitting may be prolonged from three to five or even fifteen minutes. After the galvanic treatment has been successfully applied for a week or two, the process of regeneration can be greatly aided by also employing the faradic battery, care being taken at first to use a very gentle current, so that the patient barely perceives it, applying it for only three minutes, and increasing the length of the sittings and the strength of the current very cautiously. In some cases even where galvanism is well borne, faradism will not agree with the patient, and in such a case galvanism alone should be used. As the nerves become less irritable, the sittings with both currents may be prolonged, and in some instances I even allow the

patients to apply the galvanic current for a half-hour or forty-five minutes, and the faradic for the same time or even longer.

In the early stages of neuritis, when the pain is only relatively well controlled by the analgesics that have been described, a large blister applied somewhere near the affected nerve, but not upon it, will usually relieve the pain in a marvellous way for a day or two, and in a less degree the same effect will be obtained by cauterization near the affected nerve by a Paquelin cautery or a poker brought to a white heat. The cauterization should be done only by light stripes, and it is not well to cause more than a very superficial scar, because the tissues that are enervated by a diseased nerve may break down into a troublesome ulcer, as I have seen in more than one instance.

Great attention should be paid to the general health of the patient. Anæmia should be carefully searched for by listening for any anæmic murmurs in the heart and large vessels, and perhaps by a microscopic examination of the blood. If anæmia is present, iron should be used in full doses, and the best preparations for this purpose are the albuminate of iron or the peptonate, the former in doses of ʒj three times a day, and the latter in doses of gr. ij–iij three times a day; or ferratin, gr. iv–viij three times daily, in capsules. With the iron should be conjoined abundance of nitrogenized food in the form of meat and beef-tea. It sometimes will be found useful in these cases of anæmia to combine with the iron small doses of bichloride of mercury ($\frac{1}{60}$ to $\frac{1}{32}$ of a grain) three times a day (*vide* Part II., Chap. III., "Neuralgia").

If there is no anæmia, but the patient's general health is depressed, tonics of quinine and bark, 2 grains of the former three times a day, and of the latter the officinal elixir of calisaya, $\frac{1}{2}$ ounce three times a day, should be administered. Malt-extracts will usually be found to be of great use in this restoration of nutrition, and any good fresh malt-extract that is known to contain a sufficient quantity of diastase will answer the purpose. Occasionally cod-liver oil is also useful, and when the patient can take it pure, I prefer to give a reliable oil in this way; but if an emulsion is used, it should be made fresh every week or so, and kept in a cold place, best on ice. Arsenic I have not found to be of very great use in neuritis, although it often proves of surprising value in neuralgia.

Surgical procedures are never applicable to neuritis, but they sometimes come into play in the neuralgia that may be left after a neuritis, in which event the considerations *pro* and *con* are the same as in neuralgia.

MULTIPLE NEURITIS.

DEFINITION. Multiple neuritis is that form which affects different nerve-trunks or filaments throughout the body or extremities, often seemingly in a most capricious manner.

HISTORY. The first description of multiple neuritis was given by an American physician, Jackson, of Boston, in 1822. Huss, describing chronic alcoholism in 1852, described a neuritis, without,

however, being aware of the pathology of what he was describing. In 1855 Duchenne also made known similar cases under the head of "general subacute ascending spinal paralysis." In 1864 Dumenil published his first autopsy; in 1864 Eichhorst, of Berlin, had the second one; and in 1877 Joffroy published the third. In 1880 several cases with autopsies were reported by Leyden, and since then the literature has become abundant, and has been contributed to by Lancereaux, Grainger Stewart, Mills, and by Starr.

ETIOLOGY. The causes of multiple neuritis are—

Toxæmia from such poisons as alcohol, arsenic, lead, bisulphide of carbon;
Infection of such diseases as diphtheria, typhoid, typhus, variola, malaria, tuberculosis;
Cold, dampness, and overstrain;
Syphilis;
Sex;
Age;
Epidemics.

These causes are so well known that they need not be dwelt upon except those relating to the epidemics of the disease.

This epidemic type is very prevalent among the Japanese, and is called Kakke, from two Chinese words, "kaiaku," meaning legs, and "ke," a disease, and it has been known to the Chinese as far back as 200 years before Christ, although it seems to have ceased about 200 years ago. In Japan, however, according to Starr, 14 per cent. of the men serving in the army in 1877 suffered from it, and 38 per cent. in 1878. Dr. Wallace Taylor tells me that the disease has diminished since wheat has taken the place of rice in some places, from which it would seem that this article of food bore some etiological relation to the disease. Europeans are not affected by it. It prevails to some extent in the islands of the Pacific Ocean, in India, Ceylon, the west coast of the Red Sea, Borneo, New Guinea, Brazil, Cuba, and the Dutch possessions in the China Sea. In these places it is endemic, occasionally epidemic.

CLINICAL HISTORY. The symptoms of multiple neuritis consist of motor and sensory symptoms in the upper and lower extremities, the special nerves being rarely affected, and the trophic symptoms being marked. The first symptoms to appear are the sensory ones. The disease usually develops with pain of a burning kind affecting the feet and hands, and with this are associated numbness, tingling, or formication. This pain is usually moderate in degree, but occasionally becomes extremely severe. There is also at this time a singular tenderness of the nerves and muscles, and there may be some hyperæsthesia, and even, though rarely, anæsthesia. It is seldom, however, that the tactile sense is entirely impaired, and when it is, it is in areas. As the disease progresses there supervenes a paresis of the affected limbs, and the sensory impairment may become more marked, although seldom to any complete extent, there being usually only delayed transmission of pain and temperature, some dulness of the muscular sense, and, as I have already said, impairment of the

tactile sense. The motor paresis, which may have been simply felt as a sense of weariness or fatigue, passes on to actual paralysis, usually in from ten days to three weeks. Paralysis may develop, however, in a few days, and it is probable that some cases of acute ascending paralysis terminating fatally are multiple neuritis. The paralysis is very capricious in its anatomical distribution, and may affect certain filaments in one nerve-trunk and leave the others unimpaired, and thus affect a number of nerve-trunks. The paralysis is a flaccid one, the tendon-reflexes are abolished, and atrophy supervenes in the course of a few weeks. Electrical alterations are extremely variable. In some cases there is simply diminished response to galvanism and faradism; in others, complete loss of response to faradism and response to a very strong galvanic current; in others, the response is greater with the negative pole; in others, still greater with the positive pole; and in some there is an actual reaction of degeneration. All sorts of abnormal muscular deformities may result from the different forms of paralysis, but a dropped foot and a dropped wrist are the most frequent, although the claw-shaped hand and the different forms of talipes are frequently observed. As has been said, vasomotor and trophic symptoms are not so frequent as the sensory and paralytic ones. Temporary œdema in the feet and hands, coldness and cyanosis, profuse and offensive perspiration, glossy skin, temporary inflammations of joints, and bulbous fingers, are occasionally observed.

The onset is usually without fever, or this is slight, but occasionally there may be a chill and a temperature of 103° to 104° F., and this may persist for several days. In other cases the temperature may not rise more than one-half or two degrees. The pulse is usually but slightly above the normal, and it is only in the dangerous cases that it rises above 120.

The disease is somewhat more common in males than in females.

Children are rarely affected, if at all. Most cases occur between twenty and forty.

Prognosis. The prognosis in multiple neuritis is good, although I have seen a number of cases in which permanent muscular deformity had been left. Alcoholic patients in whom the general health has been shattered usually become chronic or die, although it is surprising to see the improvement that will take place in them when drinking has been stopped and the general health has been built up. I have in several instances known such a patient to be so completely paralyzed as to be helpless in bed, and yet attending to his business in the course of twelve weeks. Nevertheless, recovery is always much slower in persons who are intemperate or who have been so in the immediate past.

Diagnosis. The diseases from which multiple neuritis should be differentiated are—

Simple neuritis;
Numb fingers;
Myelitis of the anterior cornua;
Locomotor ataxia;
Acute ascending paralysis of Landry.

Simple neuritis is confined to a nerve-trunk, and the pain is not burning, but sharp and stabbing. This limitation of the paralysis and the character of the pain alone will serve to make the diagnosis.

Numb fingers is an affection that was first described by Putnam, of Boston, and is observed in women between forty and sixty, consisting of a tingling sensation in the finger-tips and fingers, or even the hand, and rendering the movements of the hand awkward, besides causing a feeling as if it were asleep. In some of these cases there may be a slight degree of anæsthesia and analgesia, but usually there are no objective changes.

In *myelitis of the anterior cornua*, the so-called essential infantile paralysis, the affection is purely a motor one, there being no sensory implication whatever, and the muscles affected are almost invariably limited to a group in one limb, generally in the leg or around the shoulder or in the upper part of the arm. The entire lack of sensory symptoms and absence of any tendency to implicate other members will usually, therefore, make the differential diagnosis.

Locomotor ataxia is a chronic disease, is characterized by sudden, atrocious, stabbing or lightning-like pains, which come in very temporary paroxysms and are seldom continuous, and there is ataxia, either preceding the pains or succeeding them; whilst the muscular strength is unimpaired, and glossiness of skin, tapering fingers, and muscular tenderness are absent. Atrophy of the optic nerve is often present.

In acute ascending paralysis the sensory symptoms are usually entirely lacking, and the paralysis is the salient symptom, beginning in the lower extremities, ascending in a few days, or even hours, to the upper extremities, and often causing bulbar symptoms. Death generally ensues in a week or two, sometimes in a day or two.

Treatment. The treatment of multiple neuritis should be at first by absolute rest in bed in the acute cases, and in the chronic ones there should be such rest for at least a period of one to four weeks. The pain can best be relieved by means of warm applications and salicylate of soda and quinine. I shall never forget the lesson that I was first taught as to the value of hot applications by seeing the comfort and ease that were given to a youth afflicted with this disease by immersion of his feet in a tub of hot water. Swathing the feet in cotton, over which in its turn should be placed a strip of gutta-percha or oil-silk, will give great comfort. At certain periods of the day, however, especially toward night, when the pain is apt to be worse, the patient may often obtain great relief by putting the feet and hands in hot water for about half an hour, and then loosely tying around them a piece of muslin soaked in a mildly alkaline solution (10 grs. bicarb. soda to 1 oz. water), with perhaps the addition of some chloroform. Gently rubbing the limbs with oil is often useful also. Salicylate of soda in doses of 2 to 3 or 5 grains, three or four times a day, with 2 or 3 grains of quinine at the same time, is generally the best treatment. I have repeatedly observed a curious fact in regard to treatment to which I have never seen attention called. In several of my patients in whom the pain could not be controlled, and in whom

the discase was progressing, removal from the locality in which they had been attacked to another would often effect a marvellous change. As my first cases were seen in a malarious, low-lying district, I supposed that there might be some malarial element at play, but I have since observed the same thing in several other cases in which the disease had commenced in a non-malarious neighborhood, so that I now look upon this as a very important matter in the treatment, at least of the cases which we see in and around New York. If there is an actual malarial history, quinine or Warburg's tincture should be used in sufficient doses. In the alcoholic cases some skill is required in the withdrawal of the alcohol, and it should always be reduced very carefully, making use of quinine as a stimulant in its place, and the doses of quinine should be proportioned to the needs of such a patient, so that in some cases as much as 5 grains of quinine may be given every two or three hours, whilst in others only 2 grains three times a day may be needed. If quinine produces any cinchonism in the doses that are necessary to adequate stimulation in place of the alcohol, conjoin with it mild doses of the bromide of potash, 10 grains three or four times a day. When the alcohol is entirely withdrawn, place the patient upon a malt liquor, either a heavy English or German beer; and after using this for some time, taper off with an alcoholic malt-extract; *i. e.*, a malt-extract in which the diastase is combined with alcohol. When the pain has passed off, galvanism of the affected muscles and faradism will be found to be extremely useful. Massage will often be of use at this stage, but it should be very gently done without any griping whatever of the muscles, bearing in mind that its object is only to facilitate the passage of blood through the muscular tissue without mechanical irritation of it. Strychnine, $\frac{1}{120}$ to $\frac{1}{60}$ grain, three times a day, will be useful in this chronic stage, and also arsenic, best in the form of Fowler's solution, two or three drops three times a day. As improvement goes on and all tenderness dies away, and the muscles begin to return to their normal bulk, Swedish movements may be employed in addition. In the cases that have had a syphilitic history, iodide of potash in large doses, with inunctions of mercury should be used.

FACIAL PARALYSIS.

Synonyms: Bell's palsy. Prosopalgia.

The symptoms of facial paralysis are an inability to shut the eye, lagophthalmus, a loss of power of the facial muscles in the affected side, so that the naso-labial fold is lost, a puffing-out of the cheeks in whistling, and sometimes a deviation of the uvula and soft palate. The statements of authors differ very much in regard to the direction in which the tongue is pointed in facial paralysis, some maintaining that it is put out straight, others that it is protruded to the sound side, and still others that it deviates to the paralyzed side. The truth seems to be, as Hitzig states, that it is generally put out straight in the lighter forms, but that in severe and protracted cases it deviates to the sound side; and if it does, and the angle of the mouth on the

paralyzed side is drawn to its proper place, the tongue will straighten. Very often, too, there is an alteration of taste on one side of the anterior portion of the tongue, which, as we have already seen (Part II., Chap. II., "Taste"), is supplied by the lingual nerve, deriving its gustatory fibres from the chorda tympani, whilst the sense of taste in the posterior portion of the tongue supplied by the glosso-pharyngeal is unaltered. Reaction of degeneration or lesser electrical changes are also usually found in the affected muscles.

The causes of facial paralysis are exposure, a so-called rheumatic diathesis, disease of the middle ear, traumata, certain febrile diseases, lesion of the nucleus of the facial in the medulla oblongata, in the fibres of the facial in the pons, or in the centre of the facial in the motor area of the cortex, and spinal lesions. The most frequent of these causes is exposure, as to a high wind, to the draught from a window, to great excesses of temperature, or to bad weather. It has been supposed that the rheumatic diathesis—as evidenced by the predisposition to rheumatism in different parts of the body—is also a cause, but although this does occur, it is not so frequent as is generally imagined. A frequent cause is disease of the middle ear, such as caries of the petrous portion of the temporal bone, or those chronic middle-ear troubles which are so frequently left as sequelæ of scarlatina or diphtheria. Trauma can also be a cause, either from a blow upon the ear, a fracture at the base, or the pressure exerted upon the infant's head in difficult or instrumental delivery. Certain febrile diseases may predispose to facial palsy, such as diphtheria, variola, typhus, scarlet fever. The lesions of the facial nucleus of the pons and the motor area producing facial paralysis need only be mentioned in this place as a reminder. Such spinal lesions as locomotor ataxia and Landry's paralysis are occasionally accompanied by a facial paralysis.

The prognosis will vary greatly, accordingly as the disease is due to a peripheral lesion or to a central one; in other words, whether it is a neuritis or a neuritis due to a central lesion. The neuritis is usually of good prognosis. The degree of reaction of degeneration will generally be a guide in the prognosis of these peripheral cases. If there is but little alteration in the reaction of the galvanic and faradic currents, the prognosis is excellent, and the affection will pass away in a few weeks. If there is a partial reaction of degeneration—*i. e.*, the faradic and galvanic response of the nerve has been greatly impaired, but not entirely lost, whilst the muscles display qualitative and quantitative alteration in the galvanic reactions as well as increased response to mechanical excitation—the prognosis is fair, and a cure will be obtained in the course of two or three months. If there is a pronounced and complete reaction of degeneration, the prognosis is unfavorable, although even in some of these cases a relative cure may be effected, leaving slight twitching and paresis in the muscles. In the cases of lesion of the nucleus of the facial, as in some cases of labio-glosso-laryngeal paralysis, the prognosis is unfavorable. In lesions of the pons the prognosis is generally unfavorable, except where the lesion is in the pons at the apparent or

superficial origin of a nerve and is only a part of an intracranial syphilis which is brought under vigorous treatment in the early stage, when the prognosis may be fair. In lesions of the motor area of the cortex implicating the centre of the facial, the prognosis will vary according as it is syphilitic or not, being fair in the specific cases that are brought under treatment in the early stage, and bad in almost all others.

FIG. 110.

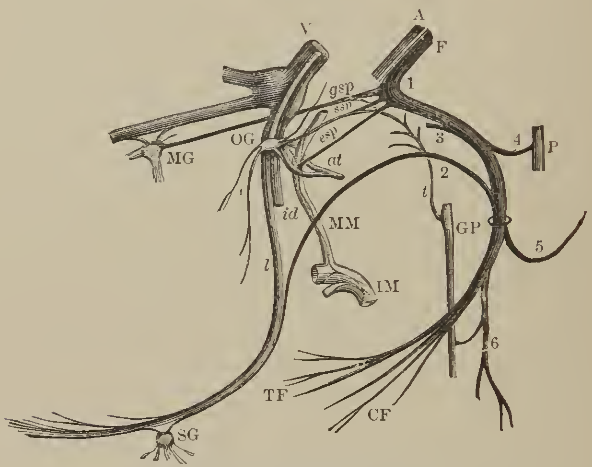


Diagram of the facial nerve, its connections and branches.
(From HERMANN'S *Physiology*.)

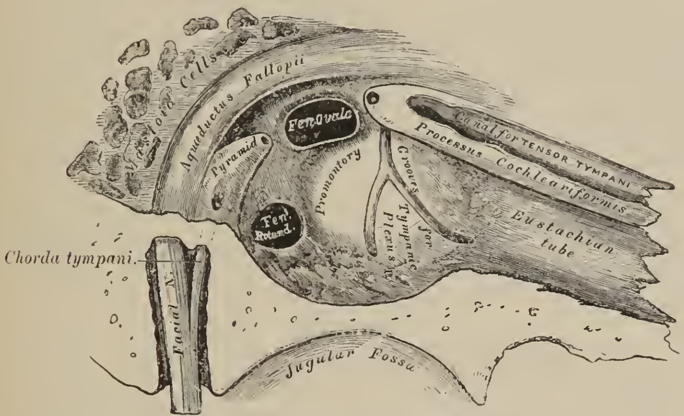
F. The facial nerve. A. Auditory nerve.
1. The geniculate ganglion.
gsp. Great superficial petrosal nerve connecting the facial and Meckel's ganglion.
ssp. Small superficial petrosal nerve connecting the facial with the Otic ganglion and with the tympanic branch of the glosso-pharyngeal.
esp. External superficial petrosal connecting the facial with the plexus on the middle meningeal artery.
2. Chorda tympani, joining lingual nerve.
3. Nerve to stapedius muscle.
4. Communicating branch with the ganglion of the root of the vagus.
5. Posterior auricular nerve.
6. Branch to the stylo-hyoid and digastric muscles.
TF. Temporo-facial division } to muscles of expression.
CF. Cervico-facial division }

V. Fifth nerve. *at*. Auriculo-temporal branch.
id. Inferior dental nerve. *l*. Lingual nerve.
MG. Meckel's ganglion.
OG. Otic ganglion.
SG. Submaxillary ganglion.
IM. Internal maxillary artery.
MM. Middle meuingeal artery.
P. Pneumogastric nerve.
GP. Glosso-pharyngeal nerve.
t. Its tympanic branch (nerve of Jacobson)

The diagnosis of a facial paralysis is a matter of no difficulty, as the open eye, the flattened face, and the cheek bellying out when any marked expiratory efforts are made, are pathognomonic. But it must

be remembered that the causative lesion may be peripheral, in the middle ear, at the base of the cerebrum, or in the nucleus of origin of the nerve in the medulla oblongata. The course of the facial in the internal capsule (p. 59), as well as the cortical centre (Plate I.), need not be considered in this process of differentiation, because a lesion of either site would not involve the fibres to the eyelid on one side, and the *levator palpebræ superioris* would not be paralyzed. A peripheral site of the lesion may be diagnosed by exclusion of one in the middle ear, the cerebrum, or the medulla. Fig. 110 will show the relation of the facial nerve to the middle ear. Fig. 111 gives an

Fig. 111.



View of the inner wall of tympanum, enlarged. (Gray.)

excellent view of the inner wall of the tympanum. From these two figures a clear idea can be obtained of the branches of the facial nerve and its connection with the middle ear. A lesion in the aqueduct of Fallopius, situated between 3 (Fig. 110), the stapedius nerve filament, and 2, the *chorda tympani*, will cause impairment of the sense of taste in the corresponding anterior two-thirds of the tongue, as well as diminished salivary secretion on the same side. If there is a lesion between the nerve-filament to the stapedius (3, Fig. 110) and the geniculate ganglion (1), there will also be abnormal acuteness of hearing—*hyperacusis*. Implication of the geniculate ganglion itself adds paralysis of the soft palate and distortion of the uvula to the symptoms. A lesion above the geniculate ganglion will cause most of these symptoms—facial paralysis, lagophthalmus, lessened saliva, paralysis of the soft palate, and deviation of the uvula; but there will be no impairment of the sense of taste in the anterior portion of the tongue, and there will probably be deafness, as the auditory nerve is close by; moreover, bone-conduction, as tested by the tuning-fork, will probably be increased (*vide* Part II., Chap. II.). A lesion at the base of the cerebrum would be certain to involve the auditory (Fig. 36), would be likely to implicate the neighboring cranial nerves, and would be caused by an intracranial neoplasm, arterial disease, intra-

cranial syphilis, cerebro-spinal meningitis, or a trauma of the skull. An isolated lesion of the facial nucleus in the medulla oblongata would be a clinical curiosity, as this is generally affected by disease involving the nuclei of other cranial nerves, such as progressive muscular atrophy, labio-glosso-laryngeal paralysis, and occasionally Landry's paralysis. Disease of the pons Varolii (Part II., Chap. I.) might also implicate the fibres of the facial. The diagnosis of a peripheral lesion of the nerve must, therefore, be arrived at by exclusion of disease in these different regions through which the facial passes in its devious career.

The treatment of Bell's palsy will resolve itself into a treatment of the lesion causing it. In the one case, therefore, the middle ear must be treated; in another, the causative lesion at the base of the cerebrum; in a third, the affection at play in the internal capsule, the centrum ovale, or the centre in the cortex; and in a fourth, the neuritis itself, although it should not be overlooked that this neuritis must also be cared for even when it is induced by one of the central lesions. A neuritis of the facial, however, is identical with a neuritis of other nerves, so far as the art of therapeutics is concerned, and my reader must, therefore, look to "Neuritis." As the latter deals mainly with the mixed nerves, though, its suggestions for the relief of pain may be disregarded. Fig. 82 will show the motor points of the facial.

NEUROMA.

Synonyms: Hyperplastic neural growths or formations. Nerve-tumors.

Neuromata are morbid growths upon the peripheral nerves. The name seems to have first been used by Odier in the year 1803. They are classified as—

Stump-neuromata.
Painful neuromata.
Nerve-trunk neuromata.
Tendril-neuromata.

Stump-neuromata are found in nerves which have been severed. The first description was given by Lower in 1669. All severed nerves have a tendency to become club-shaped at the central end, although the nerve-fibres are not otherwise altered. They are found at various times after section, from two up to many years.

The painful tubercles (*tubercula dolorosa*) are small tumors generally lying upon small subcutaneous nerves, circumscribed, movable in all directions, very painful upon pressure, and giving rise to neuralgic attacks from cutaneous irritation. They were first described by Cheselden in 1750.

Nerve-trunk neuromata are, as the name denotes, to be found upon the trunks of nerves, and are either single or multiple.

The tendril or plexiform neuromata consist of a diffuse hypertrophy of several nerve-trunks, branches, and fibrils within a certain area. The first description was given by Robin in 1854.

These different varieties are divided into two great classes: true

and false. The true neuromata consist of medullated or non-medullated nerve-fibre, and between the nerve-fibres is a varying amount of connective tissue. Of the false neuromata, fibromata are the most common, although myxomata, gliomata, sarcomata, and carcinomata are occasionally met with. Syphilomata are very rare, except on the cranial nerves.

CAUSES. The neuromata of the stump are found either in sections of nerves that have been severed, or after amputations. The age of patients suffering from painful tubercles ranges from youth up to the sixtieth year, although the majority are found between the twelfth and fortieth years. Women are more subject to them than men. Most of them are found about the leg, next around the mammæ, less frequently about the upper extremity, next in frequency about the head, and seldom in other parts of the body. The trunk-neuromata are seen most frequently from the tenth to the fortieth year, although they are observed occasionally in childhood, and even up to the eightieth year. There seems to be no difference in the susceptibility of the sexes. The optic nerve is most prone to single trunk-neuromata, and after this the median and the ulnar, next the sciatic and the radial, although they are found in almost all the nerves of the body in much less degree. Thus, contrary to what has been observed in painful tubercles, single neuromata are found about one and a half times more frequently in the upper extremities than in the lower. No explanation has ever been given of this difference. Multiple trunk-neuromata seem to occur more frequently between the twentieth and thirtieth years, but nevertheless they have been seen at all periods of life. Males are very much more subject to the disease than females. All the nerves may be affected, and there seems to be no special predisposition for any one, except that the spinal nerves are somewhat more frequently implicated than the cranial. The tendril or plexiform neuromata are often congenital, often arise in childhood, and occur up to about the thirty-seventh year. Males are twice as subject to this form as females. The head and neck are most affected. Other causes are traumata, cicatrices, and neurotic predisposition.

CLINICAL HISTORY. It not infrequently happens, curiously enough, that there are no symptoms at all, especially in the case of trunk, multiple, and plexiform neuromata. When symptoms are present, they vary in character according to the nerve that is affected. Thus, a neuroma of the optic nerve may lead to impairment of vision, of the facial nerve to paralysis, the mixed nerves to commingled sensory and motor symptoms, such as pain, anæsthesia, and muscular spasms; of the trigeminal nerve, to impairment of the muscles of mastication, or pain, or both; of the sympathetic nerve, to the various symptoms that lesions of the sympathetic give rise to; and of the pneumogastric, to gastric disturbances. When the neuromata are in superficial situations they may be felt, and they sometimes give rise to tumors that can be seen, and occasionally to bead-like swellings when there are multiple neuromata. They are sometimes sensitive

upon pressure, sometimes not, although the painful tubercles are almost always painful.

Prognosis. The prognosis of neuromata, so far as the removal of the tumor is concerned, is bad, unless it is in such a superficial position as to be reached by applications or surgical operation.

Diagnosis. The diagnosis of neuroma cannot be made unless the growth is superficial, so as to be felt or seen, although it may be suspected when there is impairment of a nerve-trunk that cannot be explained in any other way.

Treatment. In syphilitic neuromata treatment should always be tried by large doses of iodide and mercury, although I think that the mercury is better than the iodide in these cases, contrary to what has been observed in syphilis of the central nervous system. Iodide should be pushed in large doses (gtt. 60 to 200 of a saturated solution), or mercury should be pushed to the point of slight ptyalism, and it should be used in two ways, namely, by painting the neuromata, when they are superficial, with the oleate of mercury (*U. S.* and *B. P.*) once or twice daily; and also by inunction of the unguent. hydrargyri (*U. S.* and *B. P.*), ʒj night and morning. If the neuroma is single, and situated upon a nerve whose impairment of function is not of much consequence, it should be excised; thus, all neuromata of cutaneous nerves should be operated upon. Neuromata, when lying upon important nerves, however, should not be excised unless it is possible to do so without seriously impairing the function of the nerve, and sometimes this can only be determined by an exploratory incision. But there is a strong tendency to relapse, and then an operation may again become necessary. All painful tubercles should be excised. Plexiform neuromata cannot be operated on. In all cases it may be necessary to overcome the pain, and this may be done by means of analgesics employed internally. Cauterization has occasionally cured painful tubercles for the time being, although, as in the case of operation, there are often relapses; and the same may be said of counter-irritation. In some few cases cauterization has effected a cure in trunk-neuromata. In 29 cases of tendril or plexiform neuromata, gathered by Courvoisier, the results were as follows: Cured, 14; uncured, 4; relapses, 3; death by sepsis, 4; result doubtful, 2. When cauterization is performed, it should be by means of the Paquelin cautery, or iron heated to a white heat. Counter-irritation may be by means of caustic potash, or by the nitrate of silver stick.

CHAPTER IV.

DISEASES OF THE SPINAL CORD AND MEDULLA OBLONGATA.

MYELITIS.

Synonyms: Acute or general diffuse myelitis. Softening of the cord. White softening of the cord. Transverse myelitis. Inflammation of the cord.

DEFINITION. A diffuse or transverse inflammation, softening, or sclerosis of the spinal cord giving rise to commingled motor, sensory, rectal, and vesical symptoms that are different in their grouping from those of the localized or so-called *systemic* spinal diseases, such as myelitis of the anterior horns or cornua, locomotor ataxia, or lateral sclerosis.

HISTORY. The ancient physicians—among others Hippocrates, Aretæus, Galen—seem to have had some indistinct idea of myelitis, but not to have been capable of differentiating it in any way. Until the beginning of this century diseases of the spinal cord were not distinguished from diseases of its membranes, both being known by the terms of rhachialgitis or spinitis. Hildebrandt (1764–1816) introduced the term notæomyelitis. There is some little dispute as to who first coined the modern term of myelitis, Erb claiming that it was first suggested by Klohss in 1820, whilst Leyden states that it is due to Leon Hardi, although the latter included both spinal and meningeal affections under this term. The first exact description of what is termed myelitis at the present time was given by Ollivier in 1821, and Abercrombie followed in 1828 with another fair delineation of the malady—this, too, although Frank in 1792, Harless in 1814, and Klohss in 1820 had written upon the subject. Duchenne, Türck, and Romberg amplified the succeeding knowledge of the disease by their descriptions of the different forms of myelitis, both primary and secondary. About 1860 may be dated the beginning of the modern physiological, pathological, and histological study of the disease, and among the pioneers in this era the foremost has probably been Brown-Séquard, although a number of important observations have been contributed by numerous other observers, such as Oppolzer, Frommann, Mannkopf, Levy, Lockkart Clark, and Gull. More recently the school of La Salpêtrière, under the guidance of its celebrated master, Charcot, has added enormously to our knowledge of the subject, as indeed it has of many other nervous maladies; whilst a great literature has grown from the writings of a host of observers, prominent among whom are Westphal, Leyden, Erb, Seeligmuller, Strümpell, Grasset, Liebermeister, v. Renz, Gowers,

Buzzard, Ross, Stewart, Hammond, Spitzka, Seguin, Mills, Mitchell, and Wood.

Symptoms. The onset may be acute, subacute, or chronic. When acute, there may be fever up to 102° or 103°, and the pulse may be quickened, as to 120 or 130. Except in children, there are seldom any convulsions.

The symptoms will, of course, vary with the portion of the spinal cord that is affected. If reference is made to page 82, where a description is given of the different symptoms arising from lesions of the different portions of the cord, it will be seen how great may be the variety in the clinical phenomena. In all cases, however, the symptoms are of a commingled motor, sensory, rectal, and vesical nature, the particular sensory or motor area that is affected varying with the locality of the spinal cord affected by the disease. Usually the motor symptoms precede the sensory by a short interval, but it not infrequently happens that the reverse may be the case. Usually, too, the vesical symptoms succeed the distinct supervention of the motor and sensory ones, but in this case, too, it occasionally happens that the vesical symptoms are the primary ones. Whether the symptoms be motor, sensory, rectal, or vesical, they are apt to be irritative in their nature at first rather than destructive; for instance, slight disturbances of sensation and motion, or of the bladder or rectum, first occur some time before paralysis supervenes. Thus the early symptoms are apt to be some slight impairment of motion, some heavy, lead-like feeling of the limbs, which the patient may be unable for some time to distinguish from fatigue; or there may be slight sensory disturbances (*parœsthesiœ*), such as furriness and tingling in the fingers and toes, a feeling as if ants were creeping over the limbs (*formication*), or a sensation as if the fingers or toes had gone to sleep, or that peculiar sensation of a vibration running up and down the limb, which is known by the technical name of *dysœsthesia*. After a period of time, however, which will vary accordingly as the disease is acute or subacute, the irritative symptoms give place to paralytic ones. The heavy, fatigued feeling of the extremities is succeeded by a downright paralysis, and the various trifling sensory disturbances are succeeded by impairment in different degree of the senses of touch, pain, muscular sense, and temperature. There is a symptom which is peculiar to myelitis—being seldom found in the systemic spinal diseases—the *cincture* or *girdle*-feeling. It consists of a sensation about the abdomen, chest, or one of the limbs as of an encircling, constricting band or string, which will vary in breadth. This corresponds to the main focus of the lesion. Above it there is generally a zone of hyperæsthesia, answering to the irritated portion of the cord just above the focus; and this zone can be detected by the passage of a hot sponge over it, when pain will be felt instead of warmth. Paraplegia is the most common form of motor paralysis, although the upper extremities may be affected in some exceptional cases. The sensory impairment may vary from slight paræsthesia to downright anæsthesia. As a rule, paralysis does not occur so suddenly in the acute cases as in the case of myelitis of

the anterior horns, although it may be fully established within a day or two. In the subacute forms the onset of the paralysis may be spread over a week or two; whilst in the chronic cases it may be very slow for weeks or even months.

The reflexes, both cutaneous and of the tendons, vary according to the stage and localization of the disease. Reference to the chapter "General Motor and Sensory Symptoms" (Part II., Chap. II.) will make the reader acquainted with these different reflexes and the methods of eliciting them. The knee-jerk is usually diminished or abolished in early stages, but it usually becomes exaggerated later on; and in this latter period the foot-clonus is often obtainable. The cutaneous reflexes are generally lessened or lacking at first in the area of skin innervated by nerves from the affected portion of the cord, but they soon become exaggerated, too.

The electrical reactions are normal, as a rule, although some reaction of degeneration may occasionally be obtained.

The duration of the disease will vary from a few days to years. There is one rare form in which frequent relapses occur. The fact has been overlooked that some of the chronic cases, lasting several months to a year and a half, are really self-limited in their duration, and will recover with little treatment other than rest and protection from physical strain and exposure, as I have seen in several instances.

CAUSATION. The main causes of myelitis are—

Cold;
Trauma;
Muscular strain;
Violent emotions;
Acute infectious diseases;
Youth and middle age;
Sex;
Syphilis;
Disease of the kidneys;
Peripheral neuritis;
Arsenic;
Chronic lead-poisoning;
Chronic alcoholism and lepra;
Spinal meningitis;
Pregnancy.

Cold is unquestionably the most frequent of all the causes. By it is meant exposure to wet weather and storms. Feinberg produced myelitis in rabbits by refrigerating the loins with a Richardson spray-apparatus, some of the animals recovering, others dying, and in the latter a myelitic softening was found. Feinberg also produced myelitis by varnishing the skin.

We shall treat of trauma and muscular strain more especially under the heading of Traumatic Myelitis.

It is unquestionably the fact that great fright or anger is capable of aggravating, perhaps of producing a myelitis. Leyden tells of a man who died at the siege of Strasbourg from fright, who had had a stationary myelitis for years; and I myself have witnessed a death in a

case of convalescing myelitis because of the fright produced by another patient dying in the same ward.

Myelitis has been frequently observed after the acute exanthemata, typhus, acute rheumatism, puerperal fever, variola, and bacterial infection of the spleen.

Myelitis occurs generally between the ages of ten and thirty, and mostly in males.

There is no question whatever that specific infection is a large factor in the production of myelitis, although a sharp distinction must be made, for purposes of treatment, between the myelitis which occurs immediately from syphilis and that which follows a specific infection as one of its sequelæ.

As is pointed out at more length in the chapter on "Reflex Paralysis," it is a question whether the nephritic disease occasionally co-existing with myelitis is cause or effect.

While it has not yet been proven that a peripheral neuritis is capable of producing myelitis in the human being, there can be no doubt that it does so in experiments on animals, although in the latter the myelitis which results is always purulent, whilst pus rarely forms during the same malady in the human being.

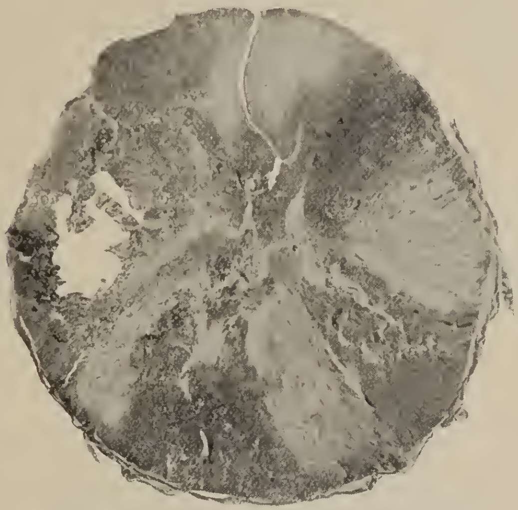
Pathology. To the naked eye the cord does not usually seem much altered, unless the softening is very pronounced or the proliferation of connective tissue has been attended with a shrinkage in volume. The cord, however, may be so soft as to be actually diffluent; or, on the other hand, greatly shrunken and hardened. Of late years the hardening-methods have been greatly improved, and it is very seldom indeed that a cord cannot be prepared for section fit for microscopic examination. Microscopically the cord may present one of several forms of molecular alteration. There may be:

1. An inflammation, generally accompanied by softening;
2. Softening, without inflammation;
3. Proliferation of the interstitial connective tissue, so-called sclerosis.

In general terms it may be said that the inflammation and softening are in proportion to the acuteness of the disease, and the interstitial connective-tissue proliferation in proportion to the chronicity. But it must not be forgotten that all three processes—inflammation, softening, sclerosis—may go on simultaneously; indeed, small areas of inflammatory softening are occasionally surrounded by a connective-tissue proliferation, and in almost all cases, except the very acute ones, inflammation, with or without softening, is found simultaneously with connective-tissue proliferation. The softening-process is usually divided into three stages, the red, the yellow, and the gray. The differences in color are due simply to the amount of blood extravasated, to the amount of fat produced, and to the amount of connective-tissue proliferation. It must be borne in mind that the elements which are affected in any disease of the spinal cord are the arterioles and venules, the nerve-fibres, the nerve-cells, the connective tissue of the gray and the white matter, and the membranes. It must also be remembered that spinal diseases are usually

attended by the formation of certain new elements, such as the so-called Deiters' cells, the granular fatty masses, and starchy bodies (*corpora amylacea*);[1] nor must it be forgotten that cysts and cavities may be formed as the result of myelitic disease. In the stage of so-called red softening the vessels are found to be distended with blood-corpuscles, frequently surrounded by a gelatinous exudation, and burst in places; the nerve-fibres may be swollen and disintegrated in axis-cylinder and medullary sheath; the nerve-cells may be swollen, broken up, presenting great vacuoles or collections of vacuoles, their protoplasmic processes destroyed or distorted in shape; there is usually a large number of Deiters' cells and fatty granular masses, whilst the pia mater is often involved in an extension peripherally

FIG. 112.



Section of the cord in a case of myelitis, showing the focus of the lesion on the left. Magnified 10 diameters.

of the myelitic process. In the stage of yellow softening the hemorrhagic and hyperæmic appearances are far less marked, having given place to a greater abundance of fatty matter. In the stage of gray softening the hyperæmia and the fattiness have largely disappeared, whilst the proliferation of connective tissue has become quite marked. Pus seldom forms, but I have seen it in two cases of purulent meningo-myelitis due to the muscular strain of a roller-skating match. The gray matter is a part that is most affected, as a rule, probably because of its greater vascularity, and the tendency to extension in this area is lengthwise, up and down. Leyden maintains that the milder forms have a tendency to dissemination. The lower two-thirds of the dorsal cord are most often affected, although the myelitis may affect any portion. Besides the central form

[1] These corpora amylacea have of late years been shown to be normal products.

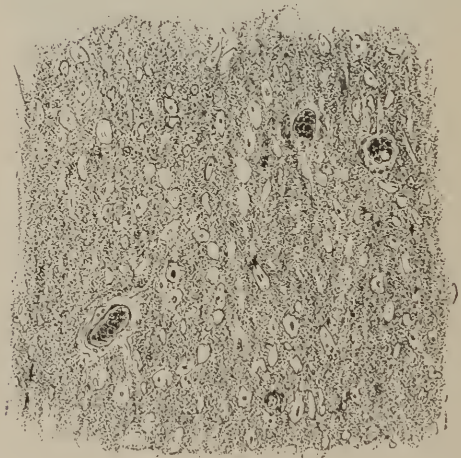
located in the gray matter, the myelitis may be diffused on the periphery of the cord or it may be one-sided, disseminated (especially after variola, as has been described by Westphal) or around the central canal (peri-ependymal).

FIG. 113.

Section of the cord in the dorsal region just above the focus of the lesion, as represented in the last figure. Magnified 10 diameters.

The termination of the myelitic process may be in—1, regeneration of the tissues; 2, sclerosis; 3, cicatrization; 4, the formation of cysts.

FIG. 114.

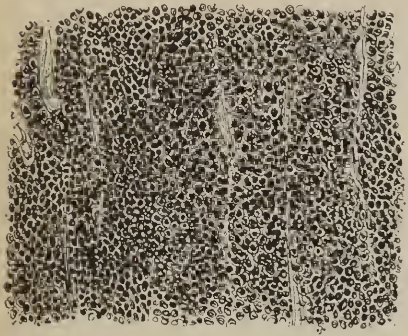


Section of the cord in a case of myelitis, showing proliferation of connective tissue and loss of nerve-fibres. Magnified 350 diameters.

Figs. 112 to 122 will give a good idea pictorially, I think, of the changes that take place in a case of myelitis. Fig. 112 is a photomicrograph of a section of the cord of a case of myelitis in my prac-

tice that commenced subacutely and terminated fatally after a duration of some ten months. In it is seen, as only a photo-micrograph can represent, the initial focus of the lesion upon the left-hand side in the lateral column, causing there a gap of substance in the section

FIG. 115.



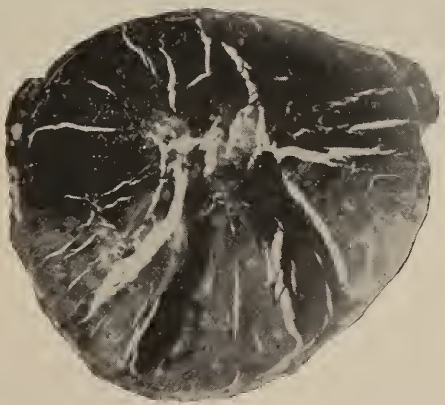
Normal cord, showing the relative proportion of nerve-fibres and connective tissue. Magnified 360 diameters.

FIG. 116.

Neuroglia of a normal cord, lateral columns. Magnified 360 diameters.

which was not due to hardening-methods, as it could be followed up and down the cord for some distance. The distorted shape of the cord can be observed, the losses of substance in the posterior columns, and the sclerotic inflammatory changes that have taken place in the anterior and lateral columns. Let me say again that none of these

FIG. 117.



Section of the cord in a case of myelitis, dorsal region. Magnified 10 diameters.

gaps of substance were artificial, for the reason that they could be followed up for some distance in the cord, and that they were surrounded by evidences of inflammatory action in the portions of the cord in which they were found. In Fig. 113 is seen a drawing of

the dorsal region of the cord above the focus of the lesion represented in the last figure. The white areas represent the places in which there has been great loss of nerve-fibres, inflammatory changes, and proliferation of the connective tissue. In Fig. 114 is seen a drawing, magnified 350 diameters, of these sclerotic portions of the cord, and if this is contrasted with Fig. 115, which represents a drawing of the normal cord in the same region, under about the same magnifying power, and with Fig. 116, which is a drawing of a portion of the neuroglia in the lateral column of the cord, magnified 360 diameters, it can easily be seen how great has been the destruction of the nerve-fibres, how great has been the increase of the connective tissue, and how swollen the vessels are and thickened in their coats. Fig. 117 is a photo-micrograph of about the same region of the cord as Fig. 113, showing as only a photo-micrograph can, the distortion of the cord and the way in which it is broken under section, although the gaps which are here so marked do not represent real losses of substance, it should be remembered, but are only due to the tearing of the cord apart under the microtone. Fig. 118 is another photo-micrograph of the same cord in the cervical region, showing the fragility of the latter, as it has also torn under the microtome, and outlining by the white lines the widespread area of loss of nerve-tubules, increase of connective tissue, and inflammation.

Fig. 118.

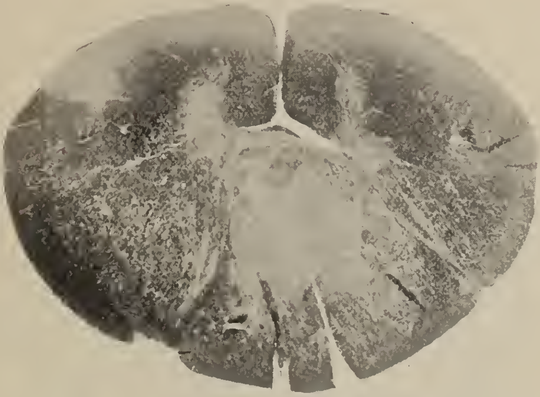
Cervical region of the cord in a case of myelitis. Magnified 7.5 diameters.

Fig. 119 is another photo-micrograph of the cord at about the same region, representing a loss of substance in the posterior columns, which, however, is magnified in the photograph, and which was increased somewhat by section under the microtome.

In the ensuing figures, 120 and 121, are represented the secondary degenerations which ensued in this case. It is a well-known law of spinal pathology that from a focus of any lesion there is apt to be a descending and an ascending degeneration, the latter being along the course of some motor set of fibres, the former along the course of some sensory set of fibres; in other words, these degenerations are

usually in the course of the nerve-impulses, so that in the motor columns they are downward as the motor impulses pass from within outward, and in the sensory columns they are upward as the sensory impulses pass from without inward. This law of degeneration, how-

FIG. 119.



Cervical region of the cord in a case of myelitis. Magnified 7.5 diameters.

ever, is by no means invariable, although it is so general as to make it an excellent guide in tracking the course of fibres and determining whether they are sensory or motor. In this case with which we

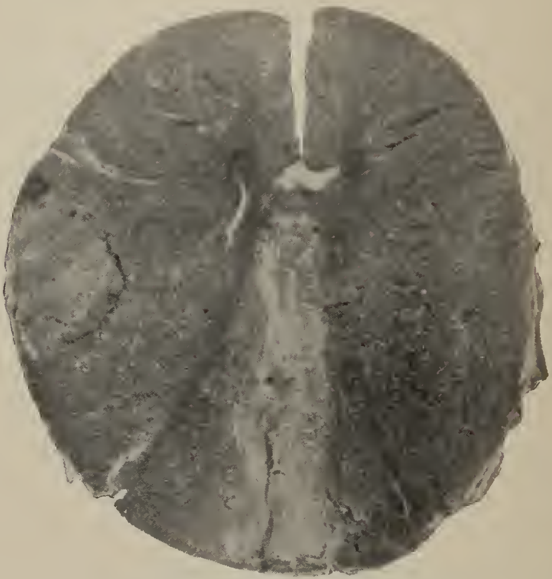
FIG. 120.

Section of the cord showing a secondary degeneration of the columns of Goll after myelitis. Magnified 10 diameters.

are dealing, however, the degeneration did not strictly follow this law, as in some places the degeneration was upward in the motor columns and downward in the sensory, although the main degenerations were in accordance with the rule. This seeming contradiction

was due to the fact that, in the case here photographed, the inflammatory changes followed nerve-strands either upward or downward, whilst the alterations in the nerve-fibres that were due to their being

FIG. 121.



Section of the cord showing secondary degeneration of the columns of Goll after myelitis. Magnified 10 diameters.

FIG. 122.

Section of the cord showing secondary degeneration of the lateral pyramidal column after myelitis. Magnified 10 diameters.

cut off from the cells of the gray matter that supplied them with nervous force (or *innervated* them) followed the usual course, and were of the usual ascending and descending type. As the posterior root-fibres split into ascending and descending branches on entering the cord (see Figs. 3 and 12 and p. 31), this also accounts for a portion of the degeneration in the two directions.

In Fig. 120, for example, a drawing magnified 10 diameters, from a section above the initial focus of myelitis, there is beautifully marked an ascending degeneration strictly limited to the columns of Goll, and marking out perfectly the peculiar wedge-shape of this fibre-system, represented in white in Fig. 121, which is a photo-micrograph of almost the same portion of the cord. This latter figure represents, as only a photograph can do, the appearance under a low magnifying-power.

In Fig. 119, which is a photo-micrograph below the initial focus of myelitis, is seen beautifully marked a descending degeneration, mainly of the lateral pyramidal columns, although, as will be observed, it extends into the lateral fundamental columns. At the right side of this figure it can be observed how the direct cerebellar column has been spared, which does not show as well upon the left side owing to the imperfection of the section. All these figures should be compared with Figs. 1 and 2, which show the normal columns of the cord.

Prognosis. Cases of very acute myelitis are usually fatal, whilst the subacute and chronic forms will depend largely as to their outcome upon the circumstances of the patient and the treatment. In every one of the two latter classes the individual case itself should be a study, and the prognosis should be based upon a number of factors, such as—

Age;
The severity and acuteness of the onset;
The progress of the malady, especially in an upward direction;
The localization of the disease in the cord;
The presence or absence of bedsores;
Sphincter-paralysis and cystitis;
The general health;
The causation;
The habits and race of the individual;
The mental state.

Whilst cases of myelitis generally occur between ten and thirty years of age, still they may be met with at an early age or at an advanced one, and in the latter case this would exert a very unfavorable influence.

A case that begins with a widespread paralysis, quickly supervening, is of very unfavorable omen, as has been said.

A rapidly extending myelitis, especially in an upward direction into the cervical centres of respiration, producing dyspnœa and cyanosis, would be almost invariably fatal. The same is true of a primarily localized myelitis in the same portion of the cord. The

lower dorsal cord can be affected most favorably as regards danger to life.

The early supervention of a bedsore within the first week is usually a very bad omen, though not necessarily so. But still worse is the formation of a large and rapidly spreading bedsore in the later periods of the disease.

Complete and enduring sphincter-paralysis is usually a bad sign, and so is also an obstinate cystitis.

It will, of course, be readily understood that the prognosis would be seriously affected by alcoholic or opium-eating habits. I have also frequently noticed that the lower orders of Germans and Irish have less recuperative power in myelitis than the other races. At the same time I have often observed that the more uneducated the individual is the more prone he is to become seriously frightened upon the recognition of a myelitis, especially as the mental faculties are usually unimpaired.

A greatly depressed individual, whether from mental disease or anxiety, is not nearly so apt to make a good recovery as one who is buoyant and sanguine.

Diagnosis. The diagnosis of a myelitis is usually made with ease, although in the chronic forms care must be exercised not to overlook the causative influence of a vertebral lesion, tumor, or an aneurism. The fourfold combination of motor paralysis, impairment of sensation, rectal and vesical paralysis, oftentimes accompanied by cincture feeling, usually differentiates the disease from the other spinal maladies, especially the so-called systemic ones. The latter, it should not be forgotten, are all chronic, with the single exception of myelitis of the anterior horns.

The diseases from which myelitis is to be distinguished are—

- The systemic spinal diseases;
- Meningitis;
- Landry's paralysis;
- Compression-myelitis;
- Concussion-myelitis;
- Neuritis, peripheral and multiple;
- Functional paræsthesia;
- Hysteria;
- Reflex paralysis;
- Myelitis of the medulla oblongata or pons, involving the lemniscus;
- Spinal tumor.

The systemic diseases are locomotor ataxia, lateral sclerosis, progressive muscular atrophy, and myelitis of the anterior horns. The first three are chronic, and the fourth is the only one which may be acute. Myelitis may be distinguished from these by attention to the following points: In locomotor ataxia there are stabbing- and lightning-pains, which are very severe, very sudden, and seldom come twice in the same place; there is the peculiar ataxia, the atrophy of the optic nerve, the Argyll-Robertson pupil, and the tendon-reflex is often absent. In lateral sclerosis there is a peculiar

spastic gait, the exaggerated tendon-reflexes, the foot-clonus, and the hasty micturition, whilst sensory symptoms and rectal and vesical paralysis are lacking. In progressive muscular atrophy there is the slow and progressive atrophy of different muscular groups, without any sensory, rectal, or vesical symptoms. In myelitis of the anterior horn there is first the motor paralysis, succeeded by the atrophy of the paralyzed muscles and the reaction of degeneration, whilst there are no sensory, rectal, or vesical symptoms whatsoever.

The most common form of meningitis of the spinal cord is that which is seen in the cerebro-spinal endemics or epidemics, and in them the mental symptoms, the coma, the implication of the cranial nerves, the retraction of the head, will make the diagnosis clear. In some cases the cerebral meningitis may be lacking, and the disease may be entirely confined to the spinal membranes, when the diagnosis may be difficult to make, unless there is considerable pain and spasm. Occasionally a purulent meningo-myelitis is set up from great muscular strain, as I have seen in two cases of roller-skaters who had attempted to win a match and who had been in incessant and outrageous motion for days. In such an event the causation and the severity of the symptoms—pain, spasm, high fever, possible delirium or coma—make the diagnosis easy.

In Landry's paralysis the onset is rapid, extending over a few hours or days; the extension is equally rapid, so that the disease quickly implicates the trunk and the upper extremities after beginning in the lower limbs, the medulla oblongata is usually affected in some degree, the termination is generally in death; and there are no sensory, rectal, or vesical symptoms, or very slight ones.

In compression producing myelitis, the compression can be detected, being either a fracture, a tumor, Pott's disease, or an aneurism; and the myelitic symptoms will be mainly localized at the portion of the cord where the compression is; besides, there will be a confused commingling of motor and sensory symptoms and exaggerated reflexes that do not correspond to the ordinary picture of a myelitis.

In myelitis resulting from concussion there will be a history of the concussion upon which the myelitis has supervened within such a length of time as to render probable the relation of cause and effect sought for. (*Vide* Chap. "Railway Injuries, etc.")

Simple neuritis of a peripheral nerve should not lead to any mistake, because the pain, the motor paralysis, and the slight and glossy-skinned œdema would all be in the distribution of the particular nerve affected. But it is not so easy always at the start to make a distinction between myelitis and multiple neuritis, although a few days' observation should settle the matter. In multiple neuritis the pains are always the primary symptom, and are usually very intense; while in myelitis impairment of sensation, such as paræsthesia and anæsthesia, are infinitely more frequent than pain; indeed, pain is seldom met with in myelitis except there is also a meningitis. In multiple neuritis the motor paralysis does not usually supervene until after several days or a week of constantly increasing and often intense pain; whereas in myelitis such a distinct

separation in point of time between pain and motor paralysis never occurs.

There is a form of functional paræsthesia that comes and goes for months and even years, which an incautious observer might confound with myelitis. The lithæmic paræsthesias—tingling, furriness, and numbness in the fingers and toes—are attended by the other nervous symptoms of lithæmia, such as tinnitus aurium, vertigo, fulness of the head, gouty or rheumatic habit, etc., and the absolute lack of motor or sensory paralysis. (*Vide* Chap. IX., "Neurasthenia.")

A hysterical woman might mislead a person for some time, especially if she be intelligent and have a motive for deception, as is not infrequently the case where actions for damages are brought. In such an event the sensations must be carefully tested, the urine must be examined for evidences of cystitis, and the subjective symptoms must be carefully weighed against those which are objective. The subject is treated at more length in Chap. IX., "Hysteria."

Reflex paralysis never produces myelitis, as I have elsewhere stated. (Chap. "Reflex Paralysis.")

In spinal tumor there is a chronic course, the symptoms are mainly localized at the level of the tumor, intense pain is present, often burning and radiating, and there is a marked immobility of the vertebral column at some point, the latter symptom never being observed in myelitis.

Treatment. The objects of treatment should be somewhat different, accordingly as the disease is of the acute or chronic type. In the former the spinal cord should be put at absolute rest, and the inflammation should be checked as much as possible. Unfortunately there are no known means of treating the softening. In the chronic cases restriction of the expenditure of energy well within the limit of fatigue, together with the use of internal or external remedies that will favorably affect the molecular changes in the cord, will make up the treatment.

In the acute cases the patient should be put upon a water- or air-bed, and care must be taken that the water is kept at such a temperature as will not chill. Ergot should be administered, either in the form of a reliable fluid extract, ʒj–ij, three or four times daily, or ergotine, gr. ij–v, three times daily. Belladonna should also be used, preferably by means of belladonna-plaster, unless a bedsore is forming; or atropia sulph. gr. $\frac{1}{100}$, two to four times daily, or a reliable fluid extract, gtt. j–ij, twice or thrice a day. Ice should also be applied along the spinal column, and the best way of doing this is to have an oil-silk bag made about two inches broad and of sufficient length, and pack this with ice. It should be applied about ten minutes daily, the patient lying on the abdomen. For the first three or four nights quin. sulph., gr. v, should be given at bedtime, preferably in capsules or tablet triturates, both freshly made, together with potas. bromide, gr. x–xx; this will often be found to have a most favorable effect in checking the progress of the disease.

In the subacute or chronic cases rest is always an important aid.

It will be found best usually to put the patient absolutely to bed for a week or two at first, then let him up for the latter half of the day, and so gradually permit him to get about again. Where this amount of rest may not be practicable or feasible, it may suffice to have the patient lie down several hours every afternoon, or to have a business man go to and from his place of business in a carriage, and stay in bed on returning home. Besides rest, numerous remedies may be employed externally and internally, as follows: Ergot may be useful in the cases of recent origin, even although the symptoms are very subacute, and in such cases it should always be tried. The same is true of belladonna, although in a less degree. The doses should be as they have been given above. The iodide of potassium has been a drug of considerable value in my hands, but the dosage should be carefully determined by the presence or absence of a specific history, as well as by the relation of the specific infection to the myelitis. In myelitis that simply follows a specific infection, moderate doses of the iodide will answer every purpose, gr. x–xv three times daily. But where the myelitis comes on during a period of specific infection, or subsequently to a cerebral syphilis, the doses must be much larger, and should be begun with grs. xx three times a day and increased quickly until symptoms of iodism supervene, or until the myelitis begins to yield. In old specific cases that have become accustomed to large doses of the iodide perfectly enormous doses may be required; thus, in one case of mine the symptoms did not improve until 700 grs. daily were reached. In all such cases the saturated solution of the iodide should be employed, each drop containing about $\frac{3}{4}$ gr., and it should be administered in a full tumbler of ice-water, Vichy, or the Bohemian Giesshuebler. As is more fully explained elsewhere (Chap. VIII., "Syphilis of the Nervous System"), I have no confidence whatever in the use of mercury. Strychnine is occasionally an extremely useful remedy in the chronic stages, as first pointed out by the late Dr. J. S. Jewell, of Chicago. It should be employed in large doses, commencing with gr. $\frac{1}{60}$, and increasing gradually to $\frac{1}{24}$ or $\frac{1}{20}$, but extreme care must be taken that the toxic effect does not manifest itself in a sudden or cumulative manner, as I have seen it do. It is best administered by the mouth, in pill, solution, or tablet triturate. An occasional dose of quinine, gr. v, combined with potass. bromide, gr. xx, at bedtime, will be found to be of great use in the more chronic stages of myelitis when there is a relapse or when the disease remains stationary.

In many cases it is advisable, for the convenience of the patient and to prevent mistakes, to combine these remedies in one prescription as follows:

℞.—Ergotin. . . . . ʒj.
Strychniæ sulph. . . . . gr. j.
Quin. sulph. . . . . ʒj. M.

Ft. in pil. no. xxx. S.—One three times daily, one hour after meals.

The bromide of potash can be added to this, 5 grains for each dose, if a tablet triturate or compressed tablet is made, as can be

done nowadays by any druggist. The ice-bag, as described above, should be applied every other day for a week or two at a time. Counter-irritation along the back is occasionally of use, although its value has been very much over-estimated. Of course, it should be used with great care until all danger of the formation of bedsores is past. Dry cups or the actual cautery, the latter either a Paquelin or electric cautery, or a poker, should be used, the latter at a white heat and applied with light strokes. Electricity is, in my opinion, the most valuable agent that we have for the treatment of the sub-acute and chronic forms of myelitis, especially in the second and third week after the onset. The galvanic, faradic, and static currents should be employed. Galvanism should be applied by large electrodes, one (Fig. 67 or 68) in the neck and the other (Fig. 68 or 69) over the lumbar spine, daily or every other day. The strength should be at first 5 to 10 milliampères, and the sitting should be from three to five minutes; as the patient grows accustomed to the electricity the current-strength and the length of sitting should be prolonged until a current of 15 to 30 milliampères is attained and the sitting is prolonged to ten minutes. But the strength of the current should never be increased to such a degree as to produce any unpleasant symptoms during or after the sitting, although much more electricity can be given in myelitis than in neuritis. The faradic current should be of sufficient strength to produce gentle muscular movement, and should be applied to the different motor points of the paralyzed limbs. (*Vide* pp. 130 *et seq.*) The static current will often be of use as an alternative with the faradic, and the one will thus frequently stimulate the flagging nerve or muscle which has failed to respond to the other. There is considerable difference of opinion as to the value of the different natural springs of Europe in their effect upon myelitis, and the reader who is desirous of informing himself upon this subject had better consult some of the foreign authors. In this country I have found that hot sulphur springs will very decidedly benefit some chronic cases, but, as they have a most deleterious effect upon others, their effect should be tested with great caution in each individual. The Turkish and Russian baths are, in my opinion, decidedly dangerous in myelitis, and I have abandoned their use. Massage is a very uncertain remedy in this disease, and should never be employed except in the chronic cases, and then empirically. It should always be given very gently and for a short time, and even then its effect can never be counted upon. Remedies should be changed from time to time in treating cases of chronic myelitis. When one procedure has lost its effect, another should be employed, and the different forms of electricity should be alternated one with the other. Change of scene and travel are useful aids to treatment in such of the chronic forms as have remained stationary after fair improvement; but any fatigue must be avoided, and especially high altitudes or regions of great barometric changes. The bedsores should be carefully attended to. The patient's position should be shifted constantly so as to avoid pressure upon any one particular spot. In sluggish cases the patient should be put on a

water- or air-bed If there be any tendency to trophic changes observed in the skin, it should be met by the application of a lotion of equal parts of alcohol and water frequently applied throughout the day; or Brown-Séquard's plan of cold water amd poultices should be tried, the cold water coming through a douche for ten minutes, and the poultice being applied for half an hour. When a bedsore has actually formed, dressing it antiseptically with a solution of bichloride of mercury, 1 part to 1000, and then coating it over with contractile collodion will not infrequently relieve small formations and hold large ones in check for some time. The cystitis which may ensue should be treated promptly by potass. citrat., gr. xxx, every night at bedtime, in a tumbler of water; or oil of gaultheria, gtt. x, in sealed capsules, just before retiring. Some very mild cases can be alleviated by the use of Vichy, Giesshuebler, or any of the other alkaline mineral waters, or the administration of gr. v–x–xv, sodæ bicarb. three times daily in a full tumbler of water. If this does not give relief, give belladonna in the fluid extract, one or two drops twice or thrice daily, and also boric acid, gr. $\frac{1}{4}$–$\frac{1}{3}$, once or twice daily in a tumbler of water. If there is much suffering, the following suppository will be found to be of value:

℞.—Ext. belladonnæ	gr. iv.	
Iodoform	gr. xxxvj.	
Opium	gr. xij.	M.

Ft. in suppositor. no. xij. S.—One every night at bedtime.

These suppositories should be made of fresh cocoa-butter, and they should have the long conical shape, and not the blunt one which is so difficult to place well up in the rectum. If there is retention of urine, soft catheters should be employed, but they must first be disinfected in a solution of bichloride of mercury (1 to 1000), and they ought to be carefully washed in this after being used. The catheter should never be used by any person who has not been shown how to introduce it properly and gently; indeed, its use should be avoided, for it is often very irritating to the bladder of a myelitic patient, and I have on several occasions seen it cause a dangerous aggravation of the cystitis. In chronic cases, grindelia robusta (fluid extract, *U. S.*), gtt. xx–xxx, once or twice daily, or the sulphate of strychnia, gr. $\frac{1}{50}$–$\frac{1}{30}$ three times daily, is often very efficient.

The constipation arising from the sphincter-paralysis should be treated by means of galvanism applied with one large electrode over the lower dorsal spine and a rectal electrode, or with the faradic current applied in the same manner; but it is far better to try to relieve it by injections of warm water and soap at first, and then, if it persists, by cascara sagrada, either the fluid extract, ʒj, once, twice, or thrice daily, or the solid extract, gr. ij–v, once or twice daily, or the so-called cascara cordial, ʒj, once or twice daily; or by the following prescription:

℞.—Ext. aloës } Ext. hyoscyami }	āā ℈j.	
Ext. nucis vomicæ	gr. v.	M.

Ft. in pil. no. xv. S.—One every night at bedtime.

MYELITIS OF THE ANTERIOR HORNS.

Synonyms: Poliomyelitis anterior. Essential paralysis of children. Infantile spinal paralysis. Atrophic paralysis of children. Acute atrophische Spinallähmung. Spinale Kinderlähmung. Paralysie atrophique.

DEFINITION. A motor paralysis, generally monoplegic, of muscles that are associated in function, followed by atrophy and electrical alterations in them; cerebral, sensory, rectal, and vesical symptoms being either absent or temporary in duration.

HISTORY. It is claimed that Underwood, in England, indistinctly recognized this disease as far back as 1784; it was better recognized by Heine, in Germany, in 1840; but the first accurate description was given by Duchenne (de Boulogne) in 1853. Charcot, in 1863, and Moritz Meyer, in 1868, made it known clinically. The first autopsy and microscopical examination were made by Gombault, in 1873, and a very thorough post-mortem examination was that of Cornil and Lépine in 1875. Since then many observations have established very decidedly the nature of the pathological changes, and the clinical symptoms have been exhaustively studied. The chronic form was first spoken of by Duchenne in 1849–50, and then more adequately demarcated in 1870; and, in later years, microscopical examinations of the central nervous system have been made by Ross, Aufrecht, Kahler, Vulpian, Provost, Archambault, Damaschino, Sinkler, and Pick.

SYMPTOMS. The disease may be acute, subacute, or chronic. It has usually three stages, the initial, the paralytic, and the chronic.

The initial stage may be febrile or non-febrile. When there is fever it is extremely variable, lasting from twenty-four to forty-eight hours, several days, or even fourteen days, being usually mild, about 102° or 103° F., although it may run as high as 110°. The pulse is usually proportionate to the fever. Other reflex phenomena, as variable as the fever itself, may accompany it, such as delirium, somnolence, convulsions, with or without loss of consciousness, muscular twitchings, vomiting, headache, temporary retention of urine, pain in the loins and in the limbs that are subsequently paralyzed, or generalized pains. Children of four or five years may be able to locate the pain, whilst younger ones will only evidence it by crying when they are touched or lifted. These febrile and other reflex disturbances are most marked in the acute form, whether in children or adults; but often in the acute form, especially in children, the paralysis may appear without any prodromata whatsoever. Thus it is by no means uncommon for a child to be put to bed apparently in perfect health, and to be found paralyzed in the morning. The non-febrile form may be accompanied or not by the other reflex disturbances. The delirium, stupor, and convulsions are generally absent in the subacute and chronic types; and in these, as in the acute forms, reflex disturbances may be absolutely lacking. One of my patients was slowly and completely paralyzed during some four weeks, in all the extremities and most of the trunk-muscles, without

the slightest symptom other than the paralysis. This initial stage will, therefore, vary from hours to weeks or months, accordingly as it is acute, subacute, or chronic.

FIG. 123.

Myelitis of the anterior cornua, showing atrophy of the leg-type on the left side.

The paralytic stage. The onset of the paralysis may be sudden or gradual. At first it is usually general, affecting most of the voluntary muscles. In the course of two or three days, however, there is a spontaneous recession of the paralysis from most of the muscles, leaving only certain groups affected. Occasionally the paralysis is limited from the first to certain muscle-groups. Usually there is a second and more gradual spontaneous recession of the paralysis during several months. In children the most common form of paralysis is monoplegia, most often of a lower extremity. In adults, paraplegia is most common, usually of the lower extremities. Still, it must be remembered steadily that any of the voluntary muscles may be affected. But all the muscles of a limb are not, as a rule, equally

implicated. In the lower extremities the flexors of the leg, the gluteal muscles, the quadriceps, and the ilio-psoas are generally spared. In the upper extremities it is the forearm- and hand-muscles that generally remain intact. The paralyzed muscles of the lower extremities are usually the tibialis anticus, or the peronei and extensors of the toes. In the upper extremities the paralyzed muscles may be the supinator longus, biceps, brachialis internus, and deltoid—the so-called "upper-arm type" of Remak or Erb; or all the muscles supplied by the radial nerve may be paralyzed, except the supinator longus. Then the

FIG. 124.

Myelitis of the anterior cornua, showing atrophy of the leg-type on the left side.

serratus anticus may be affected, together with the hinder portions of the deltoid, the infra-spinatus, and rhomboids. Thus, as will be observed, of the five lumbar segments it is usually the fourth and fifth that are the site of disease in these types; and of the eight cervical segments it is principally the fourth and fifth. Thus, analyzing 110 cases of my own, I find that the left leg is most frequently affected; next in order of frequency the right leg, then the right arm and left arm, whilst a paraplegic distribution is rare, and I have never seen a hemiplegia, although it has been observed by others. But the following table of my cases will make plain these and other details.

Total number of cases	114
Males	69
Females	45
Average age, 3 years 8 months; youngest congenital; oldest 18 years.	
Mode of onset:	
Sudden	33
With fever	51
Convulsions	9
Gradual (chronic)	3
Not stated	21
Paralysis:	
Left leg	36
Right leg	27
Left thigh	3
Left arm	9
Right arm	12
Deltoid (right)	10
Deltoid (left)	2
Paraplegia	15
Back	3
Etiology:	
Diphtheria	9
Fever	30
Convulsions	9
Trauma	12
Measles	6
Diarrhœa	6
Scarlet fever	3

Mental condition normal	114
Atrophy:	
Present	75
Not detected	2
Not stated	37
Knee-jerk:	
Lost	39
Diminished	30
Exaggerated	6
Not stated	39
Deformity:	
Talipes	15
Shortening	6
Sensory symptoms:	
Pain present	6
Limb cold	24
Tact impaired	3
Temperature subnormal	3
Reaction of degeneration complete	24
Reaction of degeneration beginning	9
Electric reactions:	
Diminished galvanic and faradic response	30
Not stated	57

FIG. 125.

Myelitis of the anterior cornua, showing atrophy of the leg-type on the left side.

During the second week atrophy of the paralyzed muscles is observed, as well as commencing alterations in the electrical response. (*Vide* p. 128.)

The paralyzed muscles are flaccid. The atrophy is demonstrable by palpation and by the change in the normal size and contour of the muscles. The electrical changes consist of diminishing response of the motor nerve trunks to faradism and galvanism, and subsequently of increasing response of the muscular nerve-filaments to galvanism, as well as of an alteration in the healthy polar formula. During the first week or ten days an increasingly strong faradic and galvanic

current must be applied to the nerve-trunks to cause muscular contraction ; and, if the myelitis of the anterior horn be severe enough,

FIG. 126.

Myelitis of the anterior cornua, showing paralysis of the leg-type in the left leg, in which the atrophy is masked by the fat of the limb, although the droop of the toes can be seen.

FIG. 127.

Myelitis of the anterior cornua, showing the upper-arm type on the left side, occurring twelve days after birth, in which there was absolute paralysis for eleven months, and complete reaction of degeneration.

it may be difficult or impossible to obtain any response to the faradic or galvanic currents. However, the electrical phenomena are different with the muscular nerve-filaments. Their response in the first week or ten days, like that of the nerve-trunks, is less and less to the galvanic and faradic currents; after this period the response to faradism continues to decrease, as in the nerve-trunks, but the response to galvanism begins to diminish—*i. e.*, a weaker and weaker galvanic current is required to produce a given muscular contraction, and the normal reaction to the poles of the galvanic battery commences to alter. We have seen that a healthy motor nerve has this polar formula[1] (page 128):

Current.	*Pole.*	*Circuit.*
1. Weak	Negative	Closing = CC
2. Stronger	Positive	Opening or Closing = AO or AC
3. Strongest	Negative	Opening = CO

From this it is apparent that the healthy motor nerve responds—by muscular contraction, of course—first and most strongly to the negative closing (CC); that the positive closing or opening (AC or AO) requires a stronger current; and that a muscular contraction at the negative opening (CO) is only seen with the strongest currents, so strong usually as to be painful. When the muscular nerve-filaments, therefore, show polar change, this is evidenced by AO or AC being obtained by the same strength of current as causes CC, or even with less; or that CO is obtained with the same current-strength as is AO, AC, or CC. All degrees of change of the healthy polar formula may be observed, of course; and it is a fair rule, with certain exception (p. 129), that the greater the polar change, the greater the structural alteration. The contraction of the affected muscles, it is also to be noted, differs from that of healthy muscular fibre of the voluntary type in being more sluggish and vermicular, thus approaching what is characteristic of the unstriped, involuntary muscles. This complex series of phenomena, consisting of diminution to faradism and galvanism in the motor nerve-trunks, and of increase to galvanism and altered polar formula in the motor-nerve muscular fibres, together with the sluggish muscular contraction, is known collectively as the *degeneration reaction* (*Entartungreaction* of the Germans) (p. 128).

The deformities which may result are the different forms of talipes known as equinus, varus, equino-varus, and calcaneus, as well as lordosis, or abnormally flexible or contracted joints. Talipes equinus and equino-varus result from paralysis of the antero-external muscular group of the leg, and are the most common. Equino-varus, when it is complicated, is caused by paralysis of the anterior group of leg-muscles, together with the adductors of the foot. Paralysis

[1] As has already been explained, the positive pole is technically called the *anode* and represented by the letter *A*; whilst the negative pole is called the *cathode*, and is indicated by *C*. The opening and the closing are denoted by *O* and *C*. But the reader should carefully read all that has been said about the diagnostic uses of electricity from page 125 onward, as these electrical reactions, that are really very simple, are apt to be sadly misunderstood.

of the calf-muscles alone, which is rare, produces talipes calcaneus. Simple paralytic talipes varus is yet more rare. The condition known as "pes cavus" is not uncommon, characterized by the hollowing of the sole of the foot and prominence of the instep, and it is attributed to paralysis of the calf-muscles and simultaneous contraction of the foot-flexors—*i. e.*, either of the long flexor of the toe, or the long peroneus. Lordosis is caused by partial paralysis of the sacro-spinal muscles. When the anterior and internal thigh-muscles are paralyzed mainly above the knee, the consequent over-action of the flexors of the leg on the thigh maintains the knee partially flexed, the leg being adducted. This condition is known as *genu recurvatum*, and is generally associated with talipes equino-varus. A joint or a limb may become so flexible as to dangle passively in any direction. There has been some difference of opinion among orthopedists as to whether the weight of the limb and the approximation of the points of insertion of antagonistic muscles have not been aiding-factors in the production of some of these deformities.

The bladder may be temporarily affected at the onset of the disease; and also the rectum, though this is more rare. Anæsthesia or retardation of sensory impressions may be temporarily observed. The paralyzed limbs are generally cold and bluish-looking, the calibre of the arteries and veins being diminished, and the temperature can always be felt to be distinctly lessened, the surface-thermometer indicating one or two degrees less than the normal. The tendon-reflex of the quadriceps is absent or diminished when the crural nerve is involved; otherwise, it may be normal or even exaggerated.

Etiology. Whilst cases occur without known cause, the causative factors are usually to be found among the following:

Age;
Sex;
Infection;
Exposure to cold and dampness;
Trauma;
Muscular or mental strain;
Multiple neuritis;
Acute diseases;
Warm weather;
Syphilis;
Menstrual suppression;
Dissipation and sexual excesses.

Whilst the disease may occur at any time of life, cases having been observed at the extremes, alternately, of three months and sixty-seven years, there are yet two periods of life which seem especially predisposed to it, viz., the first three years of infancy and the period between the eighteenth and fortieth years.

In infants both sexes seem to be equally affected, but in adults males suffer most.

There is a disposition on the part of some authors to regard the disease as infectious because several children have been affected

simultaneously in one family or in one village, and also because, as we shall learn, it is most frequent in the warm months.

Gowers has recently reported a case occurring with multiple neuritis.

It has long been observed that the onset is not infrequent after measles, scarlatina, cerebro-spinal meningitis, dysentery, diarrhœa, and it has appeared also after parturition and puerperal fever.

Of 53 cases collected by Seeligmüller, 27 were in July and August. In a record kept by Dr. Weir Mitchell for nine years, it was noted that cases became more frequent in May, June, and July, attaining the maximum frequency early in August, and rapidly decreasing in number through August, less rapidly through September, until early in October they were as infrequent as in May.

Cases have been observed during the secondary and tertiary periods of syphilis.

Prognosis. In almost all cases there is certainly left an incurable residue of paralysis. The amount of this will depend upon the severity of the disease and the thoroughness of the treatment. It should not be forgotten that the acute and subacute forms of the disease have three periods of recession: First, the spontaneous recession within a few days of the first generalized paralysis that may have been caused by the shock of an acute onset; secondly the spontaneous recession often observed in the first few months; thirdly, the recession that may be effected by early and proper treatment. All these recessions may vary in amount. On the other hand, the paralysis may be progressive in the chronic forms, and an approximate prognosis can only be framed when it is evident that the disease is not extending. After these periods have passed, the data of prognosis are—

1. The localization and degree of the paralysis;
2. The rapidity and degree of the atrophy;
3. The degree of the electrical changes in nerve and muscle, and the rapidity of their supervention.

It is very seldom that any of the vital nuclei of the medulla oblongata are sufficiently affected to cause death, and yet I have seen them dangerously implicated during the first few days. In such a case the prognosis should be very guarded. If these nuclei be not affected, the rule is that the prognosis is worse the more complete the paralysis, the more rapid the electrical changes supervene, and the more abnormal they are. The worst cases are those in which the paralysis is complete, and in which the nerve and muscle undergo in a few weeks the typical changes of the degeneration-reaction. The best cases are those in which the paralysis is incomplete, and in which there is only moderate diminution for a few weeks in the response to faradism and galvanism, without any increased galvanic excitability of the muscle or alteration in the healthy polar formula.

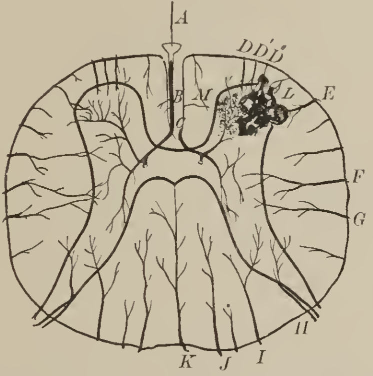
Pathology. Our views of the pathology of myelitis of the anterior horns have undergone considerable modification in the course of the last twenty-five years. The malady was first regarded as a functional one, but the autopsies and microscopical examinations

made by Cornil in 1864, Provost and Vulpian in 1867, Clark in the same year, Charcot and Joffroy in 1870, Joffroy and Parrot in the same year, and a number of others of the same kind, seemingly demonstrated that the lesion was a loss or impairment of the ganglion-cells in the anterior horns. These ganglion-cells were found to be disorganized in varying degree, so that some of them lost their processes, became smaller in size, or entirely disappeared. Of late years, however, as more autopsies have been made in cases of recent origin, it has been shown that the lesion is really a local or focal myelitis, with the usual attendants of a myelitis, such as congestion, softening, abundance of granular corpuscles, etc. The vessels are generally gorged with blood, and in a case of Archambault and Damaschino's the whole of one anterior horn was fairly flecked, like a coarse veil, with these enlarged capillaries. The lymphatic sheaths, in such cases, are filled with granular corpuscles, pressed one against the other, and containing a nucleus. Secondarily to this condition of congestion is probably the disappearance or injury of the ganglion-cells. (Figs. 7, 8, and 9.) It has been supposed that the anterior and lateral groups of cells in the anterior horns innervate the muscles concerned in the flexion and extension of joints, and that the median and lateral clusters preside over the fine muscular actions of the hands, fingers, and thumb, as well as of the ankles and toes in walking. In the course of a week or two changes are found in the anterior nerve-roots, of the nature of a secondary degeneration of nerves, also in the motor end-plates, and finally in the muscles, these neural and muscular changes being the familiar ones that have been described as occurring after neuritis (pp. 175 *et seq.*). In the cases occurring in adults the myelitis is more extended and not localized, and there is as yet considerable doubt as to whether some of the acute forms of this myelitis of the anterior horns are not identical with the so-called Landry's paralysis (pp. 224 *et seq.*). In the chronic form occurring in adults the lesions are in some cases confined to the anterior horns of the spinal cord; but it is a question as yet under discussion whether lesions entirely in the peripheral nerves may not produce the same symptoms.

Marie has recently attempted to explain the one-sidedness of the lesion by the peculiar distribution of the spinal arteries. The anterior arterial system is composed of the anterior spinal artery, which is formed by the junction in the superior cervical cord of the two ascending branches of the vertebral. This spinal artery is in front, or ventrad, of the anterior longitudinal fissure, and can be followed to the lowermost end of the cord. During its course it receives a certain number of branches from the lateral spinal arteries; and it gives off at a right-angle a series of good-sized branches which penetrate the anterior median fissure and constitute the anterior fissural arteries (Fig. 128, *B*). Each one of these penetrates the anterior cornu on one side and nourishes it with blood, but the peculiarity about each is that it does not bifurcate, but sends off, as seen in Fig. 128, only one main trunk, supplying the anterior horn on the same side. At the same time, too, this branch to the anterior horn sends off

some divisions to the neighboring white substance of the antero-lateral column. Marie believes that one of these cornual branches of the fissural artery, known as the anterior-root arteries (*DD′D″*, Fig. 128), is the focus of disturbance in acute myelitis of the anterior horn, and that this explains the one-sided distribution of the lesion. He thinks it probable that these vessels are affected either by an infectious embolism or thrombosis. It is true that there are a number of instances seeming to show that infection may sometimes cause this disease, as it occasionally occurs in limited epidemics; but the defect in Marie's theory is that it does not explain why the infection should be limited to only one vessel or set of vessels, and not extend to all the spinal arteries.

FIG. 128.



Mechanism of lesions of the anterior horn in poliomyelitis anterior.

A. Anterior spinal artery. *B.* Fissural artery. *C.* Sulco-commissural artery. *DD′D″.* Anterior root arteries. *E.* Lateral anterior artery. *F.* Median lateral artery. Lesion at *M* is from the primary branch of the sulco-commissural artery distributed to the anterior horn; whilst the hemorrhage at *L* is from one of the anterior root arteries, and it extends into the white substance of the antero-lateral column.

DIAGNOSIS. The characteristics of a myelitis of the anterior horns are the flaccid motor paralysis, which always precedes the atrophy by a distinct interval, and which is not necessarily proportionate to the atrophy; the altered electrical reactions; the infrequent and temporary implication of the nuclei of the cranial nerves; the loss of the tendon-reflex; the absence of cerebral, sensory, vesical, or rectal symptoms, except temporarily at the onset. The initial fever may render a diagnosis impossible at first from the acutely febrile affections of other origin.

The acute and subacute forms are to be differentiated from—

- Acute exanthemata;
- Meningitis, traumatic, from ear disease, or cerebro-spinal;
- Meningo-encephalitis of children;
- Neuritis, simple or multiple;
- Spinal hemorrhage;
- Transverse or diffuse myelitis;

Acute ascending paralysis;
Lead-palsy;
Progressive muscular atrophy;
Joint-atrophies;
Amyotrophic lateral sclerosis;
Hysteria.

In meningitis, traumatic, cerebro-spinal, or from ear-disease, the mental disturbance is much more marked and of longer duration; the cranial nerves may be seriously implicated, the sensory as well as the motor, which never happens in anterior myelitis; there is usually, also, the retraction of the head characteristic of meningitis; and the paralysis is almost invariably hemiplegic in distribution, whilst hemiplegia is extremely infrequent in myelitis of the anterior horn; and this hemiplegic paralysis is seldom succeeded by atrophy, except very gradually or from years of disuse.

In infantile meningo-encephalitis the acute and short fever of the onset, succeeded immediately by a paralysis, might lead to confusion were it not that, as in the forms of meningitis just described, the paralysis is invariably hemiplegic and without atrophy quickly supervening.

The ordinary neuritis of a mixed nerve, causing considerable and continuous pain, œdema, hot and glossy skin, cannot be confounded with anterior myelitis. Chronic neuritis is distinguishable by the disproportion between the pain, on the one hand, and the paralysis and atrophy on the other; often, too, by the fact that it is confined to one nerve-trunk or one branch of the nerve-trunk.

In multiple neuritis differentiation is made easy by the infectious history, in some cases the malarious neighborhood, the continuous burning-pains in both lower extremities, the steady progress within a week or two of the bilateral paralysis and atrophy, at the same time that the painful symptoms persist.

Spinal hemorrhage is usually attended by more or less persistent anæsthesia, often by marked vesical and rectal symptoms, and sometimes by bedsores. Should, however, a hemorrhage be exactly limited to the anterior horn, the differential diagnosis might be difficult.

Transverse or diffuse myelitis is indicated by such entirely distinct symptoms as marked sensory and motor paralysis, paralysis of bladder and rectum, girdle-sensation, and bedsores.

In acute ascending paralysis the rapid ascension of the paralysis from the lower to the upper limbs, then to the cranial nerves, and the usually fatal termination, are in marked contrast to the more gradual poliomyelitic paralysis, which is seldom fatal, and which is, therefore, usually followed by atrophy and the characteristic electrical changes.

In lead-palsy the exposure to lead, the characteristic line on the gums, the preceding intestinal troubles, and the characteristic "wrist-drop," will make the diagnosis easy.

In progressive muscular atrophy the atrophy precedes the paralysis, and the paralysis is always closely proportionate to the atrophy; moreover, there is a much more chronic course than in myelitis of

the anterior horn. Still, progressive muscular atrophy and chronic myelitis of the anterior horn are closely allied diseases. It was formerly thought that the distinction was that in the former the motor cells of the anterior horns were primarily affected, whilst in the latter it was the trophic cells; but this view has tacitly merged into the more rational one that when groups of cells are diseased groups of muscles are affected, whilst a lesion of single cells causes symptoms in individual muscular fibrils, especially when the cellular alterations are chronic.

In amyotrophic lateral sclerosis the paralysis and the atrophy are always accompanied by contractions and exaggerated reflexes, which are seldom present in myelitis of the anterior horn.

It is only necessary to call attention to the possibility of hysteria simulating a myelitis of the anterior horn, because the lack of the atrophy, the electrical alterations, and an unimpaired tendon-reflex will demonstrate that there is no disease of the anterior horn, even though there may be some doubt as to the existence of a motor paralysis.

Treatment. The objects of treatment should be—

1. To arrest fever and control other reflex disturbances, if there be any; 2. Possibly to affect the cellular process in the anterior horn; 3. To affect the degenerating or degenerated nerve and muscle.

The febrile disturbance is seldom sufficient to require any active treatment; but if it should be, it can readily be controlled by cold sponging or the cold pack, with or without antipyrin. The dose of this drug should vary for children from gr. j–v, once to thrice daily, in a little sweetened water. The other reflex disturbances should be treated by these same agents, and, if these are not adequate, also by the bromides and some mild opiate. In children the bromide of sodium is preferable, because it has the least tendency to upset the stomach. The dose should be from gr. v–xx, once to thrice daily. It can readily be given in simple solution in a teaspoonful or two of water. Should pain, as is very seldom the case, be so pronounced as to require an opiate, the best combination is that of the sulphate of morphia with atropia, such as is usually made in the hypodermic tablets of many of our large drug-firms. The dose in children should vary, according to the age, from atropia gr. $\frac{1}{300}$ and morph. sulph. gr. $\frac{1}{24}$ to atropia gr. $\frac{1}{200}$ and morph. sulph. gr. $\frac{1}{16}$. This had best be used hypodermically.

In the acute or subacute cases sinapisms may be applied to the spinal cord at the onset of the disease. In chronic cases a good effect will sometimes be obtained from the use of an ice-bag applied to the spinal column for ten minutes daily. This ice-bag can be made at home of a piece of oil-silk, and should be long enough to reach from the nape of the neck to the lumbar vertebræ, and about broad enough to cover the vertebral column. It must be filled with finely chopped ice. Should it produce chilliness, it should be immediately removed; and should this chilliness not disappear with its further use, it should be discontinued. I have considerable faith in the use of ergot in all cases. Its undoubted efficacy is probably due

to its action upon the dilated capillaries. The fluid extract should be used if it does not disagree with the stomach, and the doses should vary from ♏x–xxx three times daily for children, to ʒj–ʒss three times daily for adults. Its use should be persisted in for five or six weeks. Should the fluid extract nauseate the patient, ergotine may be substituted, the dose varying from gr. $\frac{1}{3}$–$\frac{1}{4}$, thrice daily, for adults. The iodide of potash is a time-honored drug that seems to have met with the approval of almost all the writers for this disease, and I am myself inclined to affirm its efficiency, more especially in the subacute or chronic stage. Moderate doses only should be given, from gr. ij three times daily in a child, to gr. x–xv in the adult. It should always be administered, to avoid the acne and the stomachic disturbances, in plenty of water, say in a wineglassful for a child and in a full tumbler for an adult; or, better still, for the latter, in a glass of Vichy or the Bohemian Giesshuebler. I am a great believer in the use of quinine in all acute or subacute affections of the anterior horn. It may be given in two ways, viz., in one or two good-sized doses, or continuously in tonic doses. In all acute cases I should strenuously advise one or two full doses as soon as the disease is recognized. In children, according to the age, gr. j–v will suffice, given at bedtime. In adults, gr. v–xv should be given in one dose at bedtime. These doses should, of course, be larger with individuals who have been accustomed to taking quinine, or with those living in very malarious districts. The dose may be repeated for one or two nights, if necessary. To lessen the possibility of cinchonism and further to control the reflex disturbances, it will be found highly useful to combine with the quinine, thus given at bedtime, a full dose of the bromide of sodium or potassium, gr. v–xxx, according to age. The quinine can be administered in capsules freshly prepared by a druggist, in the soft capsules which have lately come into vogue, or the tablet triturate. For children a very pleasant mode of administration is by means of the chocolates of tannate of quinine, of which the dose, however, should be double that of the sulphate.

The agents capable of affecting the degenerating or degenerated nerve and muscle are galvanism, faradism, massage, and gymnastic exercises. Galvanism and faradism should be employed as soon as the reflex disturbances have passed away. The galvanic current should be applied at least three times a week to the spinal column, and also to the nerve-trunks and nerve-filaments of the muscles (pp. 112–123 *et seq.*). The current should pass for three to five minutes, and should vary in strength from three milliampères for a little child to ten or twenty or even more for an adult. Faradism should be applied to the affected nerve-trunk and to the muscles themselves in sufficient strength to cause a general contraction unless the reaction to faradism is so diminished as to require too painful a current. Massage properly performed may be used with great advantage in the latter stage of the disease, and should not be begun until the degenerative process has reached its limit. When the treatment of the affected muscle has become useless, it may often be well to ascertain

whether gymnastic exercises cannot increase the strength of surrounding muscles, in the hope that the latter may be sufficiently developed in strength to compensate the atrophied ones, or whether some device or operation of the orthopædic surgeon may not relieve the deformity.

LANDRY'S PARALYSIS.

Synonym: Acute ascending paralysis.

This is a form of paralysis which was described by Landry in 1859, and it is characterized by motor, or almost entirely motor paralysis of the lower extremities, then of the trunk, then of the upper extremities, and then of the neck and face. The disease runs its course in the fatal cases in a period extending over a week or two, occasionally in a few days, or even more quickly. The paralysis is a flaccid one, usually attended by muscular atrophy, and generally without alteration in the electrical reaction of the nerve and muscle. There is usually, as has been said, no impairment of sensation, but occasionally there is some slight loss of the sense of touch and muscular sense. There is seldom any febrile reaction, although some cases have been reported in which the temperature has risen to 101° and 103°. The bladder and the rectum are not affected. Occasionally there is a slight œdema of the feet, profuse perspiration, albuminuria, or swelling of the pancreas. Most cases terminate fatally because of the extension of the disease upward into the medulla oblongata, causing death by failure of respiration or deglutition, with all the symptoms of ordinary bulbar paralysis. Some cases, however, are said to have recovered, and two noticeable instances have been reported by Rendu and Schwarz. In Rendu's case the disease continued to extend for eleven days, when the patient was greatly embarrassed in eating and breathing. On the thirteenth day, however, he began to improve; by the thirty-first day he was able to get out of bed, and on the sixty-first day he left the hospital. In Schwarz's case the disease reached its acme in about three weeks; an improvement began about the fortieth day, but the patient was not able to get out of bed in nearly six months; for three years afterward, however, the lower limbs remained weak. I have seen recovery in two cases of apparent Landry's disease, the recovery being complete in each case and commencing three and six weeks respectively after the onset of the disease. Fibrillary contractions are sometime observed.

The pathology of Landry's disease is somewhat of a mystery. Of late years the doctrine of its being either acute myelitis or acute multiple neuritis, generally the latter, sometimes the former, has been steadily growing. Thus Ross has analyzed 93 cases, and has reached the conclusion that they were all due to multiple neuritis, whether they were acute, subacute, or chronic. Nauwerck and Barth, Eisenlohr, Centanni, and others have reported cases confirmatory of this opinion; in addition, Centanni has detected a peculiar bacillus in the endo-neural lymph-space of almost all the peripheral nerves, although Lewis failed to discover any bacilli in the inoculation which

he made from a fatal case. Pitres and Vaillard found the gray matter of the spinal cord perfectly normal and few alterations in the spinal nerve-roots, but all the peripheral nerves were affected to such a grave extent that the myelin had almost entirely disappeared, the axis-cylinder being absent in many fibres in which nothing remained but an empty sheath of Schwann. The protoplasmic nuclei of the fibres were generally unchanged, however, and there were none of the active changes observed in the ordinary secondary or Wallerian degeneration. Iwanow found marked pathological alterations in the cord in two cases. These were limited to the region surrounding the central canal, with extension into the anterior horn, and consisted of an exudation of lymphoid bodies into the tissues, as well as of a fibrinous exudate around the vessels and nerve-fibres. There was also some impairment of the nerve-fibres and nerve-cells, and in places around the vessels some œdema, but no bacteria. Lockhart Clarke, Baumgarten, and Eisenlohr found similar conditions. Joffroy and Achard have also found multiple neuritis, but obliterating arteritis with it, and they believe that the neuritis was a complication or a secondary process. Sinkler has recently reported a case of apparent Landry's paralysis, in which death occurred twelve days after the onset, and in which there was shown to be an acute transverse myelitis. Besides these cases in which the marked evidences of disease of the spinal cord and peripheral nerve have been found, many others have been reported in which these structures were found to be perfectly normal. It is, therefore, probable that Landry's paralysis is due to acute changes in the cord and peripheral nerves from the action of some micro-organism as yet undiscovered, or else that it is an acute myelitis or an acute multiple neuritis, either idiopathic or of bacterial origin. The fact that the electrical excitability of nerve and muscle is not impaired for two or three weeks need not exclude diseases of the anterior horns or multiple neuritis, for Immermann observed a case of poliomyelitis that lasted fourteen days, in which the autopsy confirmed the diagnosis, yet the electrical reactions were normal; and Schwarz speaks of a marked multiple neuritis in which the electrical reactions were normal until the third week, when a marked reaction of degeneration ensued.

The causes of Landry's disease are exposure and the predisposing effect of such diseases as smallpox, diphtheria, and typhoid; it occurs more frequently in male than in female, and mainly between twenty and forty years of age, but it has nevertheless been observed in children and old persons.

The diagnosis is from myelitis, poliomyelitis anterior, neuritis, and paralytic rabies. It must be confessed that this diagnosis is a matter of considerable difficulty in the acute cases. So far as we know to-day, however, Landry's paralysis commences in the lower extremities, then implicates the trunk, the upper extremities, finally extends to the medulla, is usually entirely motor, the sensory symptoms being either entirely lacking or slight, the electrical reactions also lacking or slight, and the bladder and rectum unaffected, whilst muscular atrophy is either absent or is observed only in those cases which have

an unusually long duration. This group of symptoms will serve to distinguish the disease from those others which have been enumerated. It may be, however, that there are acute forms of myelitis, myelitis of the anterior horns, and multiple neuritis, in which the symptoms are identical with those of Landry's paralysis; and if this should prove to be true, the special designation of the malady will pass out of use. Until such time, however, it is well to preserve the name. From paralytic rabies the diagnosis can be made by the history of the bite of an animal, perhaps by mental symptoms which may be present, or by knowledge of the patient having been inoculated by the Pasteur method. (Chapter on "Hydrophobia, or Rabies.")

The prognosis of Landry's disease is very grave, as most cases terminate fatally, and it is exceptional for one to recover.

The treatment should be by means of the same methods that are employed in any acute affection of the cord or peripheral nerves, viz., absolute rest, ergot, iodide of potash, and sulphate of quinine. The patient should be put to bed and not allowed to rise for any purpose whatsoever. All noises should be excluded, and all persons kept from the sick-room except such as may be necessary to minister to the patient's wants. Ergot should be administered in the form of the fluid extract, one to two drachms three or four times a day, or ergotine (gr. ij–v three or four times daily) in case the fluid extract should disagree with the patient's stomach. Iodide of potash should be given in doses of ten to twenty grains three times daily, and should be well diluted in a half-tumbler of water. Belladonna should be given in the form of the fluid extract, one or two minims once or twice a day. The sulphate of quinine should be administered in tonic doses, two grains three or four times a day. Should the case not terminate fatally, the treatment should be that of multiple neuritis or myelitis; for which the reader must turn to the chapters upon these two subjects.

SPINAL HEMORRHAGE.

Synonyms: Hæmatorrhachis. Hæmato-myelia. Spinal meningeal hemorrhage.

Extravasations of blood may occur between the dura mater and the arachnoid, in the subdural space, between the dura mater and the bones, between the arachnoid and the pia mater, or into the substance of the cord. The extra-dural hemorrhage is the most frequent, that into the cord exceedingly rare. Spinal or meningeal hemorrhage may occur at all ages, and the most frequent cause is trauma, although severe and prolonged muscular exertion may induce it, and one of the worst cases I have ever seen was in an individual who had won the prize in an eight-day go-as-you-please walk; whilst both varieties are occasionally observed in many hemorrhagic diseases, such as purpura, smallpox, yellow fever, and typhoid. Aneurism is a rare cause. The main symptom in meningeal hemorrhage is usually a violent pain in the region corresponding to the hemorrhage, and radiating thence upward and downward; but this is not marked in spinal

extravasation. It is unnecessary to go into the different symptoms at the different levels of the cord, for these can best be studied in the table upon p. 82; but muscular spasm is a marked symptom in the meningeal forms, occasionally causing as general symptoms as those observed in tetanus, although the convulsive movements are usually localized.

The prognosis is very grave, and death will usually ensue in a few hours unless a surgical operation is done, although cases have been recorded in which the symptoms have decreased, the patient has passed through a period of localized meningitis, and recovered. The diagnosis is to be made by the suddenness and focal localization of the symptoms and the marked pain in meningeal hemorrhage; and in hemorrhage into the substance of the cord by the quick onset, although the recognition of the latter variety is very difficult, if not impossible, which makes it doubly fortunate that it so very seldom occurs. It is to be distinguished from meningitis, myelitis, tetanus, and strychnine-poisoning. Meningitis is much more gradual in its onset. Myelitis has no such sharp pains, and the symptoms of irritation are not so marked. In tetanus there will be trismus, and the onset will not be so sudden or the pain so severe. In strychnine-poisoning the paroxysms are much more extensive. The treatment should consist of absolute rest, venesection in robust individuals, or leeches or wet-cupping in those in whom venesection would not be justifiable, ice to the spine, ergot or ergotine, saline cathartics, and analgesics. The patient should be made to lie absolutely quiet on a water- or an air-bed, the bowels should be moved by an enema and the bladder emptied by a soft catheter. The patient should lie upon the face or the side, so as to keep the spine high. Ice should be applied throughout the length of the spinal column by means of cracked ice put into a rubber bag or into a home-made bag of oil-silk. The fluid extract of ergot should be freely used in doses of a drachm every two or three hours for the first twenty-four hours, and after that in lesser doses. If meningitis results, it should be treated in the same manner. If the symptoms progress rapidly, the vertebræ should be removed, provided the patient's strength is good, and the clot removed.

DIVERS' PARALYSIS, OR CAISSON DISEASE.

History. Attention was first called to this malady by Hamel, in 1820, later by Colladon in 1826, by Triger in 1848, and by Barella in 1861. Since that time there have been a number of articles upon the subject. The best writings have been those of Smith and Van Rensselaer.

Clinical History. The onset of the paralysis is generally sudden and accompanied by giddiness, headache, weakness, and nausea, occasionally with extreme pain in the trunk or legs, and sometimes transiently in the abdomen, together with vomiting. In some cases these are all the symptoms, but in most the lower extremities are usually paralyzed, together with the bladder and rectum. The upper extremities are seldom or never involved. The paralysis is

motor, although there is often an impairment of sensation. In some few instances there are swelling, heat, and discoloration of the tissues, occasionally suppuration. The skin is cool, of a leaden hue, and there is often cold and profuse sweat. The temperature is generally normal. Transient attacks occur, characterized by headache, vertigo, double vision, incoherent speech, occasionally coma; but all these are temporary, unless the coma foreruns death. Death may occur very suddenly, the patients may recover, or the condition may become chronic.

CAUSATION. There are three theories as to the causation of caisson disease, namely—that it is due to exhaustion and cold; that it is caused by the evolution of gases; or that it is the result of congestion. The objection to the theory of exhaustion and cold is that the atmospheric variations of many climates, especially that of many parts of the United States, do not produce a similar disease. The theory of the evolution of gas is twofold. Bouchard maintains, for instance, that the gas which is always normally present in the intestines is compressed by the increased air-pressure of the diver's apparatus, so that its volume varies inversely to the intensity of the compression —*i. e.*, the volume of the abdomen will become one-fourth if the pressure is four atmospheres, and the abdominal wall will be compressed against the vertebræ. In the compression of this abdominal wall, as by a vacuum in going into the diver's apparatus, the blood accumulates from all other parts of the body into the abdomen, producing in these other parts a great anæmia, and in the abdominal organs a corresponding hyperæmia. In going out of the diver's apparatus the normal atmospheric pressure reverses this process, and hence occur the peculiar pathological appearances of divers' paralysis. The objections to this theory are that the symptom of abdominal distention has not been observed, and that hemorrhages which would immediately result on the reversal of the atmospheric pressure are infrequent. The other gaseous theory is that there is an abnormal formation of gas in the blood. It has not, however, been shown by the experiments in support of this idea that the conditions to which divers are subjected are exactly the same as those to which the animals experimented on have had to submit. Bert, for instance, placed his animals under a pressure of from six to nine atmospheres, whereas Van Rensselaer states that workmen in the United States have never been in a pressure as high as five atmospheres. Bert, too, suddenly withdrew his atmospheric pressure, whereas the workmen in this country have the pressure gradually reduced during a period of three to eight minutes or more, and death has occurred after a pressure of two atmospheres. Nor has any air been found in the blood-vessels of any of the sixteen men on whom autopsies have been made in this country. The theory of congestion has involved several factors: First, that there was a congestion with black blood; second, that the congestion was followed by hemorrhage; third, that it was followed by acute revulsive anemia; fourth, that comparative stasis succeeded it. Analysis of the blood of people who have been submitted to different degrees of atmospheric pressure varying from

one to ten atmospheres has shown, however, that nitrogen is the only gas that increases markedly in the blood during the increased pressure, that oxygen increases very slowly, that carbonic acid gas decreases very tardily, and that even up to ten atmospheres the change is so trifling as not to be sufficient to account for the lesions observed; and these facts are fatal to the theory of the black blood as it implies a deprivation of oxygen, as well as that this deoxygenized blood, jointly with the congestion, is the cause of the lesions. The theory of a congestion followed by the evolution of gas is faulty, because, as we have already seen, there is no proof that gas is evolved. The theory of congestion followed by hemorrhage is open to the same criticism. The theory that congestion is followed by acute revulsive anæmia is met by the fact that the spinal cord is almost invariably congested. Of all the theories, therefore, the only one left is that there is congestion followed by comparative stasis. It has been shown that a sudden cessation of circulation will cause as sudden paralysis. Herter passed a curved needle through the abdominal wall in such a manner as to include the abdominal aorta and vena cava, a strong ligature being carried on this needle; and compression of the abdominal vessels by this method produced an instantaneous paraplegia. If the compression was relieved in less than three-quarters of an hour, the paralysis rapidly ceased; but if it was continued for an hour, permanent paralysis was induced. Whether stasis, however, must necessarily coexist with this condition has not been proved, nor has it been shown by any of these theories why the dorsal region of the cord should be the particular portion which is affected. Predisposing causes are obesity, old age, alcoholic excesses, heart and kidney disease, or lowered vitality of the body from any cause whatsoever.

Pathology. In the cases of sudden death the cord has appearances of softening, and occasionally the brain is congested, or both brain and cord may seem normal. In those cases which are fatal within a week or two after the onset of the symptoms, the spinal cord is congested or softened, and the brain is also hyperæmic. There may also be serous effusions into the spinal canal or extravasations of blood upon the dura. In the more chronic cases, lasting several weeks or months, similar appearances are found in the brain and cord. The other internal organs are also usually found congested. Microscopically the lesions are most marked in the lower dorsal region, greatest in the white matter rather than in the gray, and either of a degenerative nature or due to a parenchymatous myelitis. The greatest disease is in the posterior columns and the adjacent portion of the lateral columns. If the cases last long enough, the usual secondary degenerations are found upward in the columns of Goll and the direct cerebellar tract, and descending in the pyramidal tract. Hemorrhages are not found except upon the dura mater.

Prognosis. This is good, as a rule. The slighter attacks last from a few hours to about a week. Paralysis may continue for hours or weeks. The fatal cases are usually severe from the onset.

DIAGNOSIS. The diagnosis is very simple, and consists simply of a knowledge of the manner in which the paralysis occurred.

TREATMENT. The treatment of divers' paralysis should be that of an acute myelitis (page 225). Prevention, however, if it can be secured, would be the most useful, and those who are in charge of caissons should know certain facts that have been gained by experience. Thus, long-continued work with gradually increased pressure is not harmful. New workmen should therefore be gradually habituated to their work. The pressure should never exceed five atmospheres, and the workmen should not be allowed to work over six hours with two atmospheres, four hours with three atmospheres, and three hours with four atmospheres. The watch should be shortened correspondingly for any increase. The temperature of the air-lock will fall very rapidly during the removal of the pressure—as much as seventeen degrees in four minutes. This can be avoided. Intoxicating liquors must be forbidden. Good sleep, food, and beds should be given to the men, so that it will usually be found best to house them at the expense of the construction company. Gangs should work alternately in the compressed air and outside. Corpulent men should not be employed. The air-locks should be at the top of the shaft, so that the men may climb in the compressed air before leaving their work, or there should be an elevator. The air in the caisson should be pure. Smith suggests a rough but (he thinks) a satisfactory test for the purity of the air, consisting of a bottle of lime-water, through which the air is passed by an ordinary flexible-rubber syringe, when an excessive amount of carbonic acid will give the water a milky hue, due to the formation of the carbonate of lime. Smith also recommends that the following rules be posted up in some conspicuous place:

1. Never enter the caisson with an empty stomach.
2. Use as far as possible a meat diet, and take warm coffee freely.
3. Always put on extra clothing on coming out, and avoid exposure to cold.
4. Exercise as little as possible during the first hour after coming out, and lie down if possible.
5. Use intoxicating liquors sparingly; better not at all.
6. Take at least eight hours sleep every night.
7. See that the bowels are open every day.
8. Never enter the caisson if at all sick.
9. Report at once to the office all cases of illness, even if they occur after going home.

PROGRESSIVE MUSCULAR ATROPHY.

Synonyms: Atrophie musculaire progressive. Die progressive Muskelatrophie. Atrophia musculorum progressiva. Chronic muscular dystrophy. Myopathic atrophy.

DEFINITION. A chronic muscular atrophy and motor paralysis possessing these characteristics: The paralysis is almost always exactly proportionate to the atrophy, which is fibriliary, affects sym-

metrical muscles bilaterally, and often attacks widely separated muscles successively.

HISTORY. Our definite knowledge of this subject may be said to date from the year 1850, when Aran wrote his first article, although muscular wastings had, of course, been observed as far back as Hippocrates. In a period extending from 1853 to 1856 Cruveilhier made his first autopsies, explanatory, as he thought, of the pathology, but which have been severely criticised in later times. Duchenne followed, in 1854, with a clear-cut description of the disease in his famous book. Friedreich published a ponderous and extremely painstaking tome upon the subject in 1873. A juvenile form of the malady was described by Erb in 1884; a facial type of infancy by Landouzy and Déjérine in 1885, and a peroneal form in 1886, simultaneously by Charcot and Marie in France and Tooth in England.

SYMPTOMS. In all cases the muscular atrophy is the prominent feature of the disease, and the paralysis is always strictly proportionate to the atrophy. The characteristics of the atrophy are these: It is chronic; it affects the muscle fibre by fibre, and not by a sudden implication of the whole muscle, as in acute myelitis of the anterior horn; its progress is not generally among the contiguous muscles, but often by a jump over widely separated ones. Any of the muscles of the body may be affected. As we shall learn further on, there are cases of spinal origin, and others of peripheral causation. In the former the anterior cornua of the cord and the motor nuclei of the medulla may be affected in the same case, the progress of the disease being either downward or upward. The muscles supplied by the cranial nerves would then be affected, together with the muscles of the trunk and extremities. In the cases of peripheral origin, however, the muscles of the trunk and extremities are the ones mainly affected, the facial muscles being implicated only in children, whilst the tongue and larynx always remain intact. Through many years neurologists have demarcated many so-called "types" with the idea of associating them with pathological and prognostic differences; but no notable success has attended these attempts until within a few years. It is, however, justifiable at the present time to speak of these four groups:

1. The hand-type;
2. The juvenile type (Erb);
3. The facial type of infancy (Landouzy and Déjérine);
4. The peroneal type (Charcot and Marie, and Tooth).

1. *The hand-type.* In this the onset is generally in the right hand, occasionally in both hands. The short muscles of the thumb and the ball of the little finger are generally first affected—the so-called thenar and hypothenar eminences. It is usual for the atrophy to begin in the abductor pollicis brevis, and then be observable in the opponens and adductor. The complete atrophy of these thumb-muscles produces a condition to which the name has been given of "ape-hand" (*Affenhand*). (Fig. 129.) At about the same time, sometimes a little sooner, sometimes a little later, the interossei are implicated, as is to be seen from a sinking of the interosseus spaces

on the back of the hand and from an incomplete extension of the terminal phalanges; and when this atrophy has attained to a certain grade, it produces, together with the contraction of the antagonistic

FIG. 129.

Ape-hand.

muscles, the so-called "claw-hand" (*main-en-griffe—Klauenhand*). (Fig. 130.) Consecutively to the hand-muscles, often after a long time—sometimes even after years—the forearm or upper-arm muscles are usually attacked. In the forearm it is apt to be the abductor and extensor pollicis longus, the supinators, and the flexors. In the

FIG. 130.

Claw-hand.

upper arm the deltoid is almost invariably the first to waste, then the biceps, and finally the triceps last of all. But sooner or later the muscles of the trunk may be implicated, especially the trapezius, the pectorals, the rhomboids, and the latissimus dorsi. Atrophy is infrequent of the cervical or respiratory muscles, or of the diaphragm. After many years of duration, the bulbar nuclei may be affected, and then may ensue atrophy of the tongue, difficulty of deglutition, and death may result from inanition or respiratory disturbance.

2. *The juvenile type* (Erb). This form was first described by Erb, in 1884, and it differs essentially from the hand-type in the location of its onset, which is almost always in the muscles of the shoulder and upper arm, far less often in the muscles of the pelvis and lower extremities, whilst, as has just been explained, the hand-type primarily affects the hand-muscles, and only secondarily, often after a long time, are the shoulder- and arm-muscles involved. The shoulder- and upper-arm muscles most often atrophied are: the pectorals, trapezius, rhomboids, serrati, latissimus dorsi, longissimus, sacrolumbalis, forearm-flexors, supinator longus, triceps; and in the lower extremities, the glutæi, quadriceps, peronei, tibialis anticus. But this juvenile type of Erb is not so distinct as are the hand and infantile types.

3. *The facial type of infancy* (Landouzy and Déjérine). This was first pointed out in 1885. It generally begins with an atrophy of the muscles of expression. The face is in repose, the lips protruding, the brow like ivory, there is a seeming protrusion of the eyes; in efforts at mimicry the smile is sad, the movements of the lips are incomplete, and the immobility of the lips is in marked contrast to the animation of the eyes. (Fig. 131.) When the face has wasted, it is

FIG. 131.

Progressive muscular atrophy, facial type.

the rule that the muscles of the shoulder and arms are involved next; but certain muscles usually remain intact, such as the supra-spinalis, the infra-spinalis, the infra-scapularis, and the flexors of the hand and fingers. Exophthalmus is occasionally seen in this form, as in Fig. 131.

4. *The peroneal type* (Charcot and Marie, and Tooth). As stated above, the French authors described this form in 1866; whilst Tooth claims that he also called attention to it at the same time. As is indicated by the name which I have given it, it first shows itself in the muscles of the leg; invading the hand and then the forearm several years afterward. (Figs. 132 and 133.) Hoffman and Sachs have shown that this type is characterized by its heredity, its bilateral

implication of the leg- and foot-muscles, leading to double club-foot, the late preservation of the reflexes in it, its frequent vasomotor

FIG. 132.

Progressive muscular atrophy, peroneal type.

FIG. 133.

Progressive muscular atrophy, peroneal type.

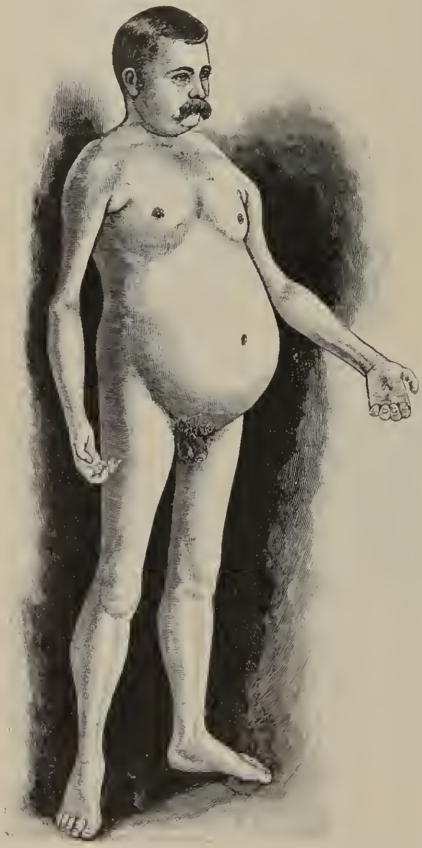
symptoms, and its occasional tendency to affect the upper extremities, even causing the claw-hand.

Hand-type.	Juvenile type.	Infantile facial type.	Peroneal type.
Muscles of thumb and fingers: Abductor pollicis brevis. Opponens. Adductor. Interossei. Lumbricales. (Ape-hand, claw-hand.) Arm-muscles: Abductor and extensor. Longus pollicis. Supinators. Flexors. Deltoid. Biceps. Triceps.	Muscles of shoulder and upper arm: Pectorals. Trapezius. Rhomboids. Serrati. Latissimus. Longissimus. Sacro-lumbalis. Flexors of forearm. Supinator longus. Triceps. Muscles of lower extremity: Glutæi. Quadriceps. Peronei. Tibialis anticus.	Facial muscles of expression. In shoulder- and arm-muscles these are intact: Supra-spinalis. Infra-spinalis. Flexors of hand and fingers.	Muscles of the leg, then the hand and forearm.

The preceding table shows, side by side, the location of the atrophy in each of these four types.

Of these four types, it is generally believed that the hand-type represents the spinal form of the disease, the juvenile and infantile facial types the muscular form, whilst the pathology of the peroneal types is undetermined. But it must be carefully remembered that

FIG. 134.



Generalized progressive muscular atrophy. Front view.

FIG. 135.

Generalized progressive muscular atrophy. Rear view.

these statements are only true in a general way, and that our present knowledge of the disease is insufficient to enable us to base a positive diagnosis as to the peripheral or central origin upon the muscular localization of the atrophy. Indeed, it is impossible to classify many of our cases coming to the clinics under any one of these four types, so general is the atrophy, frequently commingled with true or pseudo-hypertrophy, as in the remarkable case represented in Figs. 134 and

135. This classification becomes additionally difficult at the beginning of the disease in many cases, as in Fig. 136, where the supra-scapular atrophy belongs to no type.

The course of the disease is gradual, and the duration is from five to thirty years. The chronic and fibrillary atrophy may not be detected for some time, until enough of the muscle has been affected to alter the contour and consistence, which latter is then found, upon palpation, to be softer and less resilient than in a healthy muscle. The paralysis is usually proportionate to the atrophy, as has been already stated. It not infrequently happens that the loss of muscular strength first attracts the attention of the patient, or

FIG. 136.

Supra-scapular progressive muscular atrophy.

those interested in the patient, and then an examination brings the atrophy to view. Fibrillary contractions are frequently present in all the types. These consist of minute movements of the muscular fibres, and vary in extent from an occasional whipcord-like movement under the skin, likened by patients to a pulse-beat, to a wide-spread series of fibrillary movements that may set a whole muscle or muscular group into tremulous movement. The electrical alterations may be those of the reaction of degeneration, or those of the so-called "partial degeneration" (page 128), or the electrical reactions may be normal. If the researches of Gessler upon the motor end-plates of lizards and guinea-pigs should be applicable

to human beings, the reaction of degeneration will be in exact ratio to the amount of atrophy of muscular fibre. Until within a very recent time it has been supposed that a spinal origin was indicated by the reaction of degeneration; but this has also been demonstrated in some recent cases in which careful post-mortem examination could detect no disease of the nervous system.

PATHOLOGY. Since the first description of the disease by Aran in 1850, professional opinion has undergone many changes regarding its exact pathological nature. The early French school strenuously maintained that the lesion was in the ganglion-cells of the anterior gray matter of the cord and medulla; whilst Friedreich's monumental monograph in 1873 was one ponderous argument in favor of the muscular origin of the malady. At the present day it can be positively stated that progressive muscular atrophy may be of threefold origin:

1. It may be due to lesion of the ganglion-cells in the anterior gray matter of the cord and medulla;
2. It may be caused by muscular degeneration alone;
3. It may come from simultaneous degeneration of the peripheral nerves and the muscles.

The essential change in the anterior gray matter is in the ganglion-cells. These undergo various degrees of alteration. They may be totally destroyed, so that either no vestige of them is visible to a high magnifying power, or else only a formless mass of protoplasm is seen in their place; or some nucleoli or nuclei may persist; or some cells may have lost all or several of their protoplasmic prolongations; or the number of the cells may be lessened. Roger has shown that the ganglion-cells may undergo serious change or even disappear after inoculation of the micro-organism of erysipelas. Throughout the spinal cord the anterior horn may have shrunken in volume to an extent that will be visible to the naked eye, and the medulla oblongata may also be diminished in size; not infrequently, however, neither cord nor medulla is of abnormal size, and the lesions are only demonstrable by the microscope. The connective tissue, both in the spinal and medullary gray matter, is often proliferated; it may occasionally happen that the gray matter of the anterior horn or bulbar nucleus becomes converted into a dense fibrillary mass. The vessels often become enlarged and thickened in their outer tunics, the intima being seldom affected. The cranial and peripheral nerves usually undergo marked changes when their parent gray matter becomes affected. To the naked eye they usually appear thinner than normal, more transparent, often grayish or whitish in hue. Microscopically, their medullary layer is seen to be more or less affected in its continuity and consistence, in one case having broken into irregular masses or oil-globules, in another case being split irregularly in places. The axis-cylinder may be affected also in varying degree. The affected muscles may display an atrophy alone, or an atrophy mixed with hypertrophy. So far as our present knowledge can enable us to judge, the cases of spinal origin are very largely, if not entirely, manifested by atrophy alone of the primitive

muscular fibre; whilst the cases of muscular origin, especially in children, present a mixture of atrophy and hypertrophy of the primitive muscular fibres. Pseudo-hypertrophy—*i. e.*, a fatty accumulation in the sheath of the muscular fibril—may be an accompaniment of both classes of cases. Simple atrophy of the primitive muscular fibre consists of a diminution in size without loss of the transverse stripping; whilst hypertrophy is an increase in its size. The sheath of the fibre, too—the perimysium—is usually thickened, its nuclei increased in size and number. So-called "vacuoles" have also been observed, as well as collections of what appear to be giant-cells, consisting of remains of muscular and sarcolemmar nuclei that have invaded the true muscular substance.

CAUSATION. The most frequent causes of progressive muscular atrophy are—

Heredity;
Muscular strain;
Trauma;
Acute diseases;
Age;
Continuous cold;
Lower classes;
Sex;
Lead-poisoning;
Syphilis.

The juvenile and infantile facial types are very largely hereditary, the heredity sometimes extending in the most remarkable manner through several generations. The hand and peroneal types are only occasionally hereditary.

There can be no doubt that, next to heredity, muscular strain is the most potent cause of progressive muscular atrophy. Friedreich narrates case after case of this kind, and my experience has tallied with his. It is for this reason that the right upper extremity is most prone to be affected, especially in those who use this member much in manual labor.

Trauma needs only to be mentioned, because of its possible medico-legal bearings. I have seen several cases caused, in all probability, by injuries sustained in railroad accidents, and other forms of concussion of brain and cord may act in the same manner.

Progressive muscular atrophy has been often observed to follow typhus, diphtheria, measles, acute rheumatism, parturition, and cholera; so that these acute diseases must have some unfavorable influence upon the cells of the muscles and the anterior horns.

Although certain types, as we have seen, may appear at certain periods of life, the disease may yet occur at any age, so that this factor is of relative subordination.

Several cases have been observed, both by myself and others, of the induction of the disease by prolonged or continuous exposure to cold of the part afterward affected. I have seen this especially in market-men working in refrigerators, and in ice-dealers.

It is singular that cases of progressive muscular atrophy are seldom

encountered among the rich or well-to-do classes of our population, except where there is an hereditary causation. Its frequency in the lower classes is probably due to the greater use of the muscles by them and to the greater exposure.

Males are most frequently affected, probably because of their greater exposure to the various causes.

Lead-poisoning and syphilis are both infrequent causes.

DIAGNOSIS. The diseases with which progressive muscular atrophy is liable to be confounded are—

Chronic myelitis of the anterior horn;
Peripheral neuritis, simple or multiple;
Syringomyelitis;
Atrophic joint-affections;
Amyotrophic lateral sclerosis;
Muscular pseudo-hypertrophy.

In chronic myelitis of the anterior horns the motor paralysis is primary, the atrophy is secondary and in proportion to the paralysis, the spread of the disease is from muscle to muscle, not from fibre to fibre, and contiguous muscles are usually affected; whilst in progressive muscular atrophy the atrophy is primary, the paralysis is secondary, the paralysis is strictly proportionate to the atrophy, the atrophy spreads from fibre to fibre, and widely separated muscles are involved.

The diagnosis in simple neuritis of a nerve-trunk is easy when a mixed nerve is affected, and when such symptoms are present as marked pain in the distribution of the affected nerve, glossy and slightly œdematous skin, heat of the surface, and sensory impairment. But confusion may arise in neuritis of a mixed nerve when, as is not infrequently observed, the motor fibres are almost entirely affected. In such cases, however, the paralysis will be strictly limited to the distribution of the nerve and will be primary, whilst the atrophy will be secondary; and the spread of the disease will be within the distribution of that nerve, and not by jumps to widely separated muscles. In multiple neuritis the onset is often acute, and even if it is chronic the burning pains and the sensory impairment will make the diagnosis easy. Besides, the endemic, epidemic, alcoholic, or arsenical causation may be apparent; and in any event the atrophy is not primary, nor does the disease spread fibrillarily from muscle to muscle.

The diagnosis can usually be made between progressive muscular atrophy and syringomyelitis. My reasons for this belief are elaborately set forth in "Syringomyelitis," and I must ask the reader to peruse this carefully.

In atrophic joint-affections the atrophy is around the joint, and it is plainly to be seen that the articular disease has been primary and the atrophy secondary.

In amyotrophic lateral sclerosis the atrophy and paralysis are always accompanied by contractures and exaggerated tendon-reflexes to a far greater extent than is found at any time in progressive muscular atrophy.

In any case of progressive muscular atrophy there may be a certain amount of muscular pseudo-hypertrophy; but this is limited, and in no case has it the characteristics of the disease to which the name *per se* has been given of muscular pseudo-hypertrophy. In this latter affection the hypertrophy is in the lower extremities, generally contrasting sharply with the atrophy of the upper extremities. It is only in the earlier stages of pseudo-hypertrophy—when it is first noticed that the child is defective in its movements—that any confusion should arise.

Prognosis. The prognosis of all forms of progressive muscular atrophy is unfavorable, although some seeming cures have been reported in cases that were apparently of peripheral origin.

Treatment. The treatment should be by means of galvanism, faradism, massage, rest, cod-liver oil, arsenic, strychnine hypodermically, generous diet, and occasionally surgical or orthopædic treatment.

The galvanic current should be applied to the spinal column, and also to the nerve supplying the affected muscles, both to the nerve-trunks and to the muscular nerve-filaments (pp. 124, 125, 126). The strength of current should vary from five to twenty or thirty milliampères, according to the effect upon the particular patient, as some will do best with the lesser quantity, others with the greater; but the painful susceptibility of neuritis (*vide* p. 199) is never seen in this disease. The sittings should be from five to ten minutes, two or three times a week. The faradic current should be applied to the motor nerve-trunks and muscular nerve-filaments of the affected muscles, and it should be strong enough to be somewhat unpleasantly felt by the patient. Both currents should be used in accordance with the principles that have been laid down. Gentle massage of the affected muscles should be given two or three times a week, and with it the muscles will often be seen to increase slowly in bulk and strength. Rest of the affected muscles to the extent of not straining them should always be observed. I am no believer in gymnastic exercises in this disease. Cod-liver oil should always be employed in cases of progressive muscular atrophy, unless there is a tendency to pseudo-hypertrophy. Some sweet, fresh oil should be used, preferably pure; but if an emulsion must be employed, this should be prepared anew every few days in warm weather and every week in cold, and even then kept in the ice-chest. Arsenic will be found to be a useful alterative in this malady, and may be given in the form of liquor potassæ arsenitis, ♏iij–v, three times a day, well diluted in a wineglass of water, and taken after meals. If there be any indication for iron, it may be given in conjunction with this drug; and the best preparation is either ferratin, gr. iv–viij, or the dialyzed iron, ʒj–ij after meals, in a full tumbler of water, or the citrate (gr. ij–v three times daily). A generous and varied diet should always be observed. Surgical or orthopædic treatment will sometimes overcome a muscular deformity temporarily.

LOCOMOTOR ATAXIA.

History. W. Horn, in 1827, was the first approximately to describe the disease, and Stanley, in 1840, called attention to the ataxia; but Todd may be really called the discoverer of the malady itself, in 1847. Romberg, in 1851, Russell Reynolds, in 1855, W. Gull, in 1856 and 1857, followed with good descriptions of many of the symptoms; but that of Duchenne, in 1858 and 1859, was by all odds the best. Cruveilhier described the microscopic appearances of the cord correctly. Türck was the first one to describe it microscopically. Leyden and Friedreich, in 1863, Topinard and Jaccoud, in 1864, have been followed by scores of others since—notably, Hammond, Mitchell, Seguin, Spitzka, and Gray.

Causation. The causes of locomotor ataxia are:

- Syphilis;
- Sex;
- Trauma;
- Alcoholism;
- Other spinal diseases;
- Chronic ergotism;
- Chronic arsenicism;
- Heredity;
- Urban population.

Syphilis is the most frequent of all the causes of locomotor ataxia, as is now generally acknowledged, although this doctrine was met by the most furious opposition when first maintained by Fournier, in 1875. The number of cases in which an antecedent history of syphilis has been obtained varies in different writers from 30 to 90 per cent. The most remarkable confirmation of this view is to be found in the recent observation of Minor, of Moscow, of the infrequency of locomotor ataxia among the Jews, who are singularly free from syphilitic infection. I may say, in passing, that this present belief in the frequent specific origin of locomotor ataxia is a rare posthumous tribute to the clinical genius of Duchenne (of Boulogne), to whom we owe the first description of the symptoms, and who especially remarked the occasional efficacy of iodide of potash. But it must not be supposed that all the cases that have had a syphilitic history are necessarily cases of cerebro-spinal syphilis. On the contrary, it should be distinctly understood and carefully borne in mind that most cases are those in which the locomotor ataxia has developed after specific infection, and that these have an entirely different prognosis and therapeusis from those others in which the symptoms of locomotor ataxia are immediately due to cerebro-spinal syphilis.

It is most frequent in males, for what reason is unknown, although it is probable that the lesser exposure of the female sex to specific infection and other causes may account for the difference.

Trauma is undoubtedly the cause of some of the severest forms. One of the worst cases that I ever saw began shortly after a terrible railroad disaster, in which the patient fell more than a hundred feet through a railroad bridge.

Alcoholism is more apt to set up the peripheral form.

Locomotor ataxia may be conjoined with other spinal diseases and with spinal meningitis, so that its peculiar symptoms may be combined with those of these other maladies.

Chronic ergotism, as has been pointed out by Türck, may set up locomotor ataxia, as may also chronic arsenicism.

Heredity is of small importance as a causative factor, and even then only indirectly. Thus a neurotic heredity of various nervous diseases may be found in a small proportion of cases; but direct inheritance is extremely rare, except in the allied hereditary form which is known as Friedreich's disease.

According to Gowers, locomotor ataxia is more frequent in urban than in rural populations in England; but I cannot say that this is true of America.

Clinical History. The symptoms of locomotor ataxia may be divided into three classes, viz., the pathognomonic, the non-pathognomonic, and the symptoms which are merely associated with the disease.

The pathognomonic symptoms are:

Stabbing and fulgurant pains, which are vagabond, sudden, and atrocious;
Absent tendon-reflex;
Ataxia, motor and static;
The Argyll-Robertson pupil;
Atrophy of the optic nerve;
Ptosis;
Crises—gastric, laryngeal, nephritic.

It is the association of two or more of these symptoms that is absolutely necessary for the diagnosis of locomotor ataxia, and it may be said that the association of several of these symptoms is not known to occur in any other disease. The stabbing-pain is often described, with a singular identity of words, as feeling like a knife plunged into the limb and twisted around. It may be less, however, and may simply consist of a sharp pain which the patient cannot describe. The fulgurant pain is, as the name indicates, a pain somewhat resembling the electric shock passed through the limb. Both these stabbing and fulgurant pains, however, generally have three characteristics in common—they seldom can be expected in any particular spot (are, therefore, *vagabond*); they come and go very suddenly, and they are very severe—what the French have called *atrocious*. The tendon-reflex of the quadriceps extensor is almost invariably absent; but before this can be confirmed a careful examination should be made upon the bare skin with the aid of the method of "reinforcement" (p. 167). The ataxia may be both motor and static; *i. e.*, when movements are made, or when the patient is sitting or standing (*vide* p. 172). When the lower extremities are greatly affected, the patient walks with a flopping movement, bringing the heel down upon the ground like a flail; or, in a still more marked degree, the foot may be brought down upon the outer or the inner side. In the slighter degrees the patient cannot walk a

chalk-line or a crack in the floor; or, in the still slighter degrees, this may be done with the eyes open, but not with them shut. The patient's attention is often called to the ataxia for the first time by the unsteadiness on a dark night or in a dark room. Yet he can walk very well if he is allowed merely to touch the finger of some other person. If seated, the patient will fail to hit with his foot the finger of a person held before him. If the upper extremities are affected, it will be found that the patient cannot, if the eyes are shut, swing the arm up and hit the tip of the nose with the tip of the index-finger, or cannot bring the tips of the fingers of the two hands together quickly and swingingly. These phenomena are known as motor ataxia. Static ataxia is failure to co-ordinate the muscles so as to preserve the equilibrium when sitting or standing, and is evidenced by swaying and uncertain movements of the trunk and head when the person is sitting or standing. The pupil, which is known as the Argyll-Robertson pupil, has, in its typical form, three symptoms—myosis, failure of response to light, and sluggish contraction to accommodative movements. The pupils are usually minutely contracted, sometimes to such a degree as to suggest morphinism; on the other hand, they are sometimes but slightly affected. The loss of response or diminished response of the iris to light is probably due to the atrophy of the optic nerve, which is a primary atrophy, and is not accompanied by any signs of retinitis. Its white, glistening appearance is unmistakable to any tyro in ophthalmoscopy (Fig. 94). The ptosis is generally a transient symptom, and usually unaccompanied by paralysis of any external ocular muscles. The so-called crises are peculiarly violent attacks of pain in the gastric, laryngeal, or iliac regions, lasting for hours and simulating organic gastric, nephritic, or laryngeal disease.

There is no other disease in the whole range of medicine which has so original and marked a symptom-group as locomotor ataxia when it is characterized by these stabbing and fulgurant pains (which are vagabond, sudden, and atrocious), by the absent tendon-reflex, by the motor or static ataxia, by the Argyll-Robertson pupil, by the optic-nerve atrophy, by the transient ptosis, and by the peculiar crises. These, therefore, are pathognomonic symptoms.

The non-pathognomonic symptoms which may be found in locomotor ataxia, but which are often found in other diseases of the nervous system, are:

The so called Romberg-symptom;
Anæsthetic zones;
Joint-lesions;
Various sensory symptoms;
Constipation;
Vesical symptoms;
Vasomotor and trophic symptoms.

The so-called Romberg-symptom is a swaying motion of a patient standing with the eyes closed; but, as it is generally due to a loss of tactile sense on the plantar surface of the foot, sometimes to lesions in the different regions of the cerebro-spinal axis, it is not necessarily

a symptom of locomotor ataxia. Not infrequently anæsthetic zones are found in the lower extremities in locomotor ataxia, although they may be found in other forms of myelitis and in some cases of neuritis. The retardation of pain-conduction is evidenced by sticking a pin or needle into the patient and then observing that he feels it only after a perceptible interval of several seconds or it may be a minute. This is quite a common symptom of locomotor ataxia, but it is quite as common in many forms of myelitis affecting the sensory tracts. Various sensory symptoms may be observed in locomotor ataxia, as well as in other forms of myelitis. A cincture or girdle-feeling may be felt around the body, as if a cloth or band were compressing the patient round about. A prick on one limb may be referred to the other, sometimes to an identically symmetrical position (*allocheiria*); or a prick in one spot may be felt in many places on the same or on both legs (*polyæsthesia*). The positive pole of a galvanic battery may give the first sensation when the current-circuit is closed, instead of the negative pole doing so. Subjective sensations of cold, heat, tingling, pins, needles, etc., are often felt. Constipation is a very frequent symptom. The symptoms due to simultaneous disease of other portions of the cord are muscular atrophy of the muscles of the limbs or trunk, occasionally a lingual atrophy, or contracture.

Locomotor ataxia usually begins in the lower limbs, and may commence in the upper ones, although rarely, Déjérine finding one such beginning in 101 cases in the Bicêtre.

The disease is an eminently chronic one in the vast majority of cases, although acute cases do occasionally occur, running their course in a few months. The sensory symptoms are usually the first; indeed, they may last many years before the ataxia supervenes—in one case of mine some twenty-one years. But the ataxia, it must not be forgotten, is sometimes the first symptom. The course of the disease is generally slowly progressive. Long spontaneous remissions were claimed by the older writers, but it is extremely doubtful whether they did not admit many cases which have since been shown to be due to other forms of spinal disease. The extension of the disease is generally upward, so that the upper extremities become affected in time; or, if the upper extremities be first affected, the extension is downward. The ataxia may render the patient absolutely helpless, unable to rise without help, to stand alone, or even to sit without support. Usually the motor strength is in marked contrast to the helplessness of the ataxia, and I have been in the habit for years of showing my class a man who could not stand alone, but whose legs could not be flexed or extended by the strongest man in the room. Motor weakness is almost invariably the last of all the symptoms, and many patients die without having displayed it. Many cases of locomotor ataxia gradually pass into a form of dementia resembling that of general paralysis of the insane, but differing from it in the slightness of the delusions and hallucinations, in the lack of the extravagant delusions which are known as the delirium of grandeur, and also in the lack of periods of excitement. In proportion as this dementia supervenes, the ataxia diminishes, and it has been supposed that this is due to the loss of cortical control.

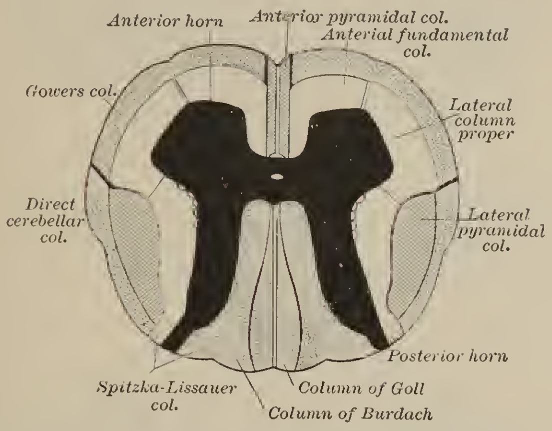
Pathology. The synonyms of locomotor ataxia, tabes dorsalis and posterior sclerosis, are misleading in regard to the real pathological lesion, since this is neither a softening nor tabes, is occasionally of cervical and not of dorsal origin, and modern research, as we shall see, has demonstrated that the pathological alterations are by no means confined to the posterior column. Until within about a decade locomotor ataxia was thought to be due to implication of the posterior columns of the cord and the adjacent gray matter of the posterior horns, and there was a difference of opinion as to whether these posterior columns and cornua were together or separately paths of sensory conduction. Then Flechsig's embryological researches mapped out the direct cerebellar column. Then Woroschiloff demonstrated that section of both posterior columns and the posterior halves of both lateral columns in the lower dorsal region of the rabbit did not prevent sensory impulses passing from the hinder limbs to the motor structures in the cervical cord and the brain. Three years afterward, in 1877, Gowers was the first to confirm anatomically what Woroschiloff had observed physiologically by the discovery of the antero-lateral peripheral tract, which has since come to be known as the Gowers column (Fig. 133), although Westphal, Kahler, Pick, Strümpell, and Leyden had also partially figured this before. That this is a sensory tract is proved by the fact that it is the site of an ascending degeneration, as is made evident by the cases of tabes dorsalis of Haddon, Gowers, Westphal, and my own, and by Tooth in compression-myelitis. The objection of Déjérine, that the degeneration of this peripheral tract might be due to meningitis, has been abundantly disproved, especially by Sherrington, who has found this column in the eighth month of uterine life, and has demonstrated also that it takes on its medullary covering at a different time from other strands of the cord. Bechterew, in his turn, has subdivided the Gowers tract into an anterior and a posterior portion, and has been confirmed by Sherrington's observations. In 1885 Spitzka called attention to a tract which he describes as along the entry-line of the posterior roots of the posterior horn on one side and the periphery of the cord on the other, giving to this the name of the analgesia tract, as he has found it impaired in all cases of analgesia, and healthy in others. According to Spitzka, there is a column of vertical fibres immediately adjoining the gelatinous head of the posterior horn, which he believes to bear the same relation to the gelatinous substance that the ascending roots of the fifth pair bear to the gelatinous substance of the tuber cinereum of Rolando, and this analogy inclines him to believe that the tract may be possessed of trophic functions, although he can affirm nothing more positive than that it was healthy in those cases in which there were trophic disturbances, as well as in that other class of cases with cutaneous lesions directly related to the fulminating-pains.

Further researches, however, have shown that there is a still further subdivision of the sensory columns, as is delineated in Figs. 1, 2, 3, and 137, and described on pages 21 *et seq.* Moreover, nowadays, the relationship of the ganglion upon the posterior root of the

sensory fibres has been very clearly shown (page 30), as well as the modes of entry of the posterior or sensory fibres into the cord, their subdivisions into ascending and descending branches, their free termination, and the origin of still other sensory fibres from the so-called column-cells of the cord (page 31). All these anatomical data have enabled us to obtain a very much clearer idea of the pathology of locomotor ataxia.

In the earlier stages of the disease the Spitzka-Lissauer column, the anterior root-zone, the middle root-zone, the column of Goll, and the median zone are implicated in the lumbar region. (See Fig. 2.)

FIG. 137.

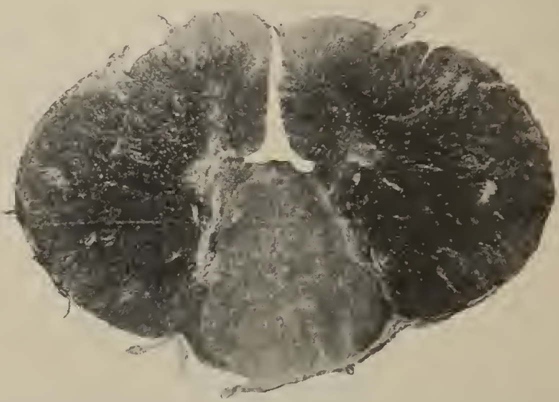


Columns of the spinal cord, according to our present knowledge.

In other words, the nerve-fibres affected are in the Spitzka-Lissauer tract, the fine fibres coming from the posterior roots, and which some authors consider to be really collaterals of the posterior roots. Whatever they be, these fibres, on entering the cord, pass in a nearly horizontal direction toward the Spitzka-Lissauer column, then they curve upward to enter the gelatinous substance. Some of them, however, continue on with the larger fibres of the posterior roots and pass partly into the gelatinous substance and partly into the centre of the posterior horn. As these fibres have but a short course in the Spitzka-Lissauer column, and soon leave it, it is easily understood why the alterations of them are principally found in the lumbar cord and, to a less extent, in the dorsal. The anterior root-zone receives a large number of its fibres directly from the posterior roots. They enter the posterior cornua and disappear in the anterior portions of this, avoiding the columns of Clark. There is reason to believe that this zone contains a certain number of commissural fibres uniting different levels of the gray matter. The middle root-zone, as has been shown in Fig. 2, has two systems of fibres. The first passes through the posterior columns, enters the columns of Clark, or, where these are not present, disappears midway between the boundary of the anterior and posterior cornua. The second system

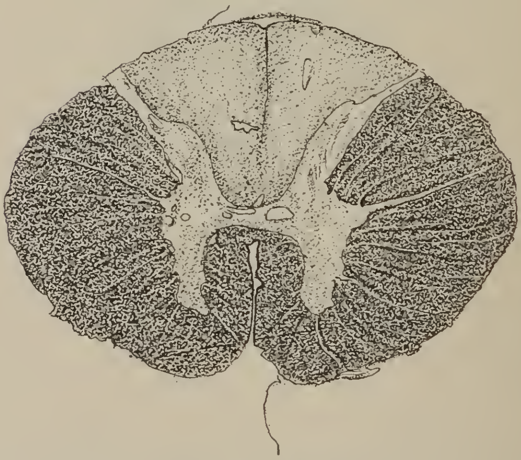
is composed of fibres which, further up, constitute by their union the column of Goll. The origin of the fibres of the median zone is not known. The column of Goll is described on page 79. In other

FIG. 138.



Section from a case of locomotor ataxia, showing degeneration of the columns of Goll and Burdach. Dorsal region. Magnified 10 diameters.

FIG. 139.



Section from a case of locomotor ataxia, showing degeneration of the columns of Goll and Burdach. Dorsal region. Magnified 10 diameters.

cases the whole of the posterior columns between the diverging posterior horns are implicated, as well as the Spitzka-Lissauer columns; and this is what is most frequently seen in the well-established cases, as in Figs. 138, 139, 140, which are sections from a case of locomotor ataxia that had lasted ten years, and was quite typical in its symptoms.

Fig. 138 is a photo-micrograph, magnified 10 diameters, of a section of the cord in the dorsal region in which the degeneration of the posterior columns (columns of Goll and Burdach) are outlined. Little

gaps are seen in the cord, which, however, are entirely due to the process of hardening.

Fig. 139 is a drawing from the lower dorsal region of the same cord magnified 10 diameters, showing very clearly the degeneration of the posterior columns (columns of Goll and Burdach) quite up to the inner side of the posterior cornua, and implicating a portion of the Spitzka-Lissauer columns.

Fig. 140 is a drawing of a section of the same cord in the cervical region, magnified 10 diameters, showing the same implication of the posterior column, but not to a like extent, as a relatively less affected mass is seen toward the posterior gray commissure and running out like a broad wedge into the posterior columns.

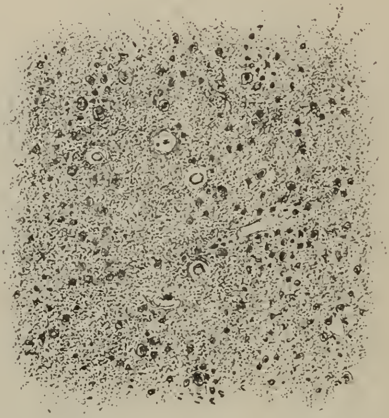
FIG. 140.

Section from a case of locomotor ataxia. Cervical region. Magnified 10 diameters.

The alterations in the column of Clark were first described in 1885 by Lissauer. As has already been shown, these columns of Clark consist of certain ganglionic cells and a network of fine nerve-fibres. In locomotor ataxia the fibres alone are affected, disappearing to a large extent, whilst the cells remain unimpaired. Lissauer is inclined to believe that the alteration in these nerve-fibres is the first pathological phenomenon in locomotor ataxia, and in 12 of 13 cases examined by him he found degeneration of the fibres extending into the column of Clark from the Spitzka-Lissauer column, and in two cases of my own degeneration was quite distinct, although I had no proof of its being in the early stage. Lissauer also states that this degeneration of nerve-fibres is more accentuated on the internal side of the column of Clark, which he explains by supposing that this receives especially the posterior root-fibres coming from the lower portions of the cord that are most apt to be diseased, whilst the external portions contain fibres coming from higher levels. This preservation of the cells in the column of Clark explains the relative infrequency of implication of the direct cerebellar column, as it is from these cells, which are true column-cells of the cord, that the fibres of the direct cerebellar tract take their origin. The central canal of the cord is occasionally affected, and the cells of the ependyma may be proliferated, occasionally to such an extent as to block the lumen; and in other cases there is a dilatation of the cord; but

it is uncertain whether this is a phenomenon of true locomotor ataxia or a coincidental occurrence of gliosis. These alterations of the nerve-fibres may be followed up to the nucleus of the column of Goll

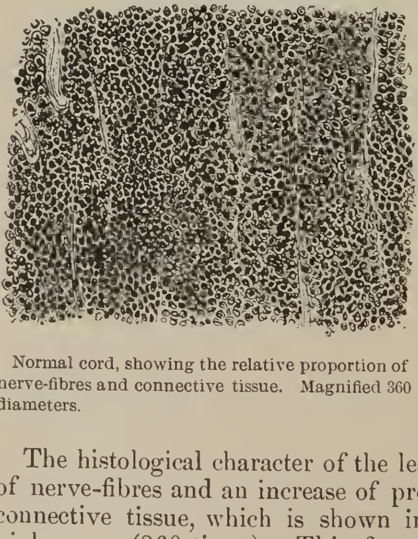
FIG. 141.



Section of a portion of the cord in locomotor ataxia. Magnified 360 diameters.

and the nucleus of the column of Burdach. In the medulla oblongata there is often found atrophy of the nuclei of the pneumogastric and the glosso-pharyngeal. In some cases the nucleus of the hypoglossal is also affected; and in others also the ascending root of the solitary fasciculus.

FIG. 142.



Normal cord, showing the relative proportion of nerve-fibres and connective tissue. Magnified 360 diameters.

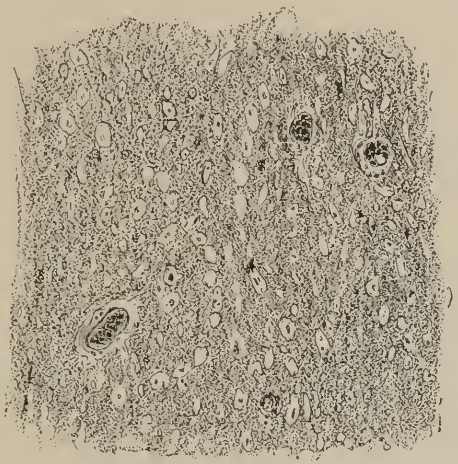
FIG. 143.

The neuroglia of a normal cord, lateral columns. Magnified 360 diameters.

The histological character of the lesion consists of a disappearance of nerve-fibres and an increase of proliferation of the interstitial or connective tissue, which is shown in Fig. 141, a drawing under a high power (360 times). This figure should be contrasted with the

connective tissue and the nerve-fibres of the normal cord, as represented in Figs. 142 and 143, as well as with the molecular alterations in myelitis, as described upon page 218 (Figs. 114 and 144).

FIG. 144.



Section of the cord in a case of myelitis, showing proliferation of connective tissue and loss of nerve-fibres. Magnified 350 diameters.

It is a question at the present day as to what is the exact nature of these changes in the nerve-fibres and the connective tissue. One view is that they are all due to a cerebro-spinal disease of unknown origin; whilst another is that they consist of secondary degenerations proceeding from disease of the cells in the ganglion upon the posterior root. There are no data, however, upon which to settle this question. Déjérine, Pitres, Vaillard, Oppenheim, Siemerling, Nonne, Westphal, and Pierret claim that the disease may be occasionally caused by a peripheral neuritis. Outside of the two cases reported by Déjérine, however, there is very great doubt as to whether peripheral neuritis really produces anything like the clinical symptoms of true locomotor ataxia; indeed, I constantly show cases in my clinic which might be taken for locomotor ataxia, but in which the distinction can readily be made, as has been shown in the chapter upon neuritis. In some cases there is a slight diminution in size of the whole cord, especially when the lesions are very pronounced. Occasionally, also, there is a slight thickening or opacity of the pia mater at the level of the posterior roots; but the relationship of this meningitis to the parenchymal lesions is unknown. In other instances the implicated columns are of a grayish tint which makes them easily distinguishable from the neighboring fibres. Finally, cases have been reported wherein the cord and periphery have been absolutely unaltered.

DIAGNOSIS. The diagnosis is a matter of ease when the full

symptom-group is present, viz., stabbing and fulgurant pains, which are sudden, vagabond, and atrocious; Argyll-Robertson pupil; absent knee-jerk; motor and static ataxia; optic nerve-atrophy; ptosis, and the so-called crises. But the diagnosis may become difficult when some of these pathognomonic symptoms are lacking. There may be said to be three cardinal symptoms, one of which must be present in order to make a diagnosis of locomotor ataxia, viz.: the peculiar pains, the peculiar ataxia, or an absent knee-jerk. Upon no one of these alone can a diagnosis be made, but any one of the three associated with other pathognomonic symptoms may make the diagnosis certain. Thus many different combinations may be present:

Pains	Pupil.
Pains	Ataxia.
Pains	Optic-nerve atrophy.
Pains	Loss of knee-jerk.
Ataxia	Loss of knee-jerk.
Ataxia	Pupil.

It must never be forgotten that pains closely simulating these stabbing and fulgurant ones are occasionally observed in transient cases, seemingly of neuralgia or neuritis, and I have known of one serious mistake made in this way. Atrophy of the optic nerve may come, it is well known, from many causes, but a primary atrophy is rare except in this disease. The gastric crises are often simulated by functional attacks of gastric neuralgia.

The diagnosis should be from—

Neuralgia, especially of the trigeminus;
Rheumatism;
Intracranial tumors;
Chronic myelitis;
Lateral sclerosis;
Disseminated sclerosis;
Hereditary ataxia;
The toxic ataxias;
Chorea;
Paralysis of the flexors of the leg;
Cerebellar disease;
Cerebro-spinal syphilis.

As has been said, neuralgia may sometimes have a peculiar stabbing or fulgurant pain, which may be severe, sudden, and vagabond, or it may be difficult to get a sufficiently exact description from the patient; in either event the other symptoms of locomotor ataxia should be sought for.

The same remarks are true of rheumatism.

Locomotor ataxia may sometimes be mistaken for intracranial tumor when the optic-nerve atrophy is observed and the pain is in the region of the trigeminus and associated with gastric crises, so that the atrophy is regarded as due to a precedent neuro-retinitis, the trigeminal pain as being of intracranial origin, and the gastric crises as being the vomiting of cerebral headache. But search for the other symptoms of locomotor ataxia will make the diagnosis plain.

A chronic myelitis may have many of the non-pathognomonic symptoms of locomotor ataxia, and may even have some of the pathognomonic ones, if the lesion extend into the proper portions of the cord. In such an event the non-pathognomonic symptoms would either be the only ones, or they would have preceded the pathognomonic ones by a distinct interval.

Lateral sclerosis, pure and simple, would have symptoms of exaggerated reflexes and contractures, and not the pathognomonic symptoms of locomotor ataxia.

In disseminated sclerosis the posterior columns and cornua might be affected as in locomotor ataxia, but then the symptoms of this latter disease would be combined with the symptoms of disseminated sclerosis, viz., tremor of the intentional type, nystagmus, and scanning speech.

Hereditary ataxia, so-called Friedreich's disease, is generally a disease of youth, and is simply the ataxia without any of the sensory symptoms. The diagnosis may sometimes be difficult, and then the reader had best consult the chapter on "Friedreich's Disease."

The toxic ataxias, due to brass, arsenical poisoning, ergotism, or copper-poisoning, may be recognized by ascertaining the occupation of the patient.

The early stage of chorea is sometimes mistaken for locomotor ataxia, because there really is an inco-ordination of movement. In every such case, however, stripping the patient will usually make plain the quickly begun and quickly ending fibrillary movements of chorea in the muscles of the trunk, head, and extremities. In the chronic and hereditary forms of chorea the differential diagnosis should be still more easily made.

In paralysis of the flexors of the leg, from neuritis or myelitis of the anterior horns, there is a peculiar flop of the leg in walking not unlike that observed in locomotor ataxia, but there are none of the pathognomonic symptoms of the latter disease.

In cerebellar disease the ataxia is of a gently swaying kind, technically known as *titubating*, rather than the peculiar flop of the locomtor ataxia. It is sometimes, however, difficult to distinguish this titubating sway from the static ataxia of locomotor ataxia, but other signs of cerebellar disease will make the diagnosis plain, viz., occipital headache, neuro-retinitis, or some paralysis.

Cerebro-spinal syphilis is often confounded with locomotor ataxia, and yet it is extremely important to make the diagnosis, inasmuch as many cases of cerebro-spinal syphilis with symptoms of locomotor ataxia have been cured, at least to all outward appearance, or at any rate are much more tractable than cases which have simply had syphilis at some period in the past. If the spinal lesion of this disease be a secondary degeneration starting from a primary focus of inflammation or cellular alterations in the lower dorsal or lumbar cord, it would seem reasonable to assume that this primary focus would be the lesion to direct treatment to, especially in the syphilitic cases, and that the secondary degenerations resulting therefrom would be as hopeless as they are in other diseases of the cord.

Fournier expressly states that the prognosis is much better in what he calls a pre-ataxic stage. If any case of locomotor ataxia with a history of antecedent syphilis has cerebral symptoms, it should be remembered that these will probably indicate intracranial syphilis, and careful examination will bring out the symptom-group to which I have called attention of late years (*vide* Chap. VIII., "Syphilis of the Nervous System"), viz., the quasi-periodical headache and the obstinate insomnia, or either present alone, ceasing upon the supervention of any convulsive or paralytic symptoms; or else there might have been some mental dulness, coma, convulsion, or symptoms indicative of a lesion at the base of the brain.

PROGNOSIS. Almost all cases of locomotor ataxia can be improved to the point of great diminution or cessation of the pains, lessening of the ataxia to a varying degree that is dependent upon its amount and duration at the beginning of treatment, and marked bettering of the general health. In other words, most cases can be very greatly improved, or even practically cured, so far as the arrest of symptoms is concerned. We do not know whether this means that the lesions in the cord have disappeared, or merely that they have not progressed as in Erb's patient, for this is the only *post-mortem* on record of an improved case.

TREATMENT. In all cases of locomotor ataxia it should be carefully ascertained as to whether or not there has been a history of syphilis, because if there has been, the iodide of potash must be used in large doses. Again and again have I seen syphilitic cases fail to respond to the other treatment, and pass on into startling results when the iodide has been used in the proper way. In cases in which the iodide does not agree well with the stomach, mercury may be used in conjunction with it. There is a difference of opinion as to the result of mercurial treatment of syphilitic cord lesions by mercury and iodide, but, if I may judge by my own experience, the iodide is always far the most reliable. The latter should be given in the saturated solution, commencing with 20 drops three times a day, given in a full tumbler of water. Sometimes it is best to administer it after meals, and sometimes immediately before, and this can be ascertained by experiment with the patient. It has been supposed that the difference is due to the effect of iodide upon the starch in the stomach. The best method of using mercury is by means of a mercurial ointment, which should be put into capsules which contain a drachm, which can then be opened by the patient, and thus there will be no danger of exceeding the dose. A drachm of the mercurial ointment once or twice a day will be sufficient in conjunction with whatever doses of iodide the patient's stomach will bear. As these cases of locomotor ataxia have usually been greatly discouraged by various therapeutic experiments, it is always best to put them to bed absolutely at first. How long they should remain there will depend upon the severity of the case. If the patient is very much exhausted or emaciated, or has a high degree of ataxia or great pain, it may be well to keep him in bed for four or five weeks absolutely. If, however, neither the pain nor the ataxia is marked, and the physical

condition is fair, a week or two will answer; but this is a question which can only be determined by the judgment of the physician. In all instances care should be taken to have too much rest rather than too little. Whatever the period of rest be, great caution should be exercised in putting the patient again upon his feet. This should only be done at first for a half hour or an hour a day, and should consist merely of allowing him to sit up during that time, instructing him to lie down immediately when he feels tired. The pulse should also be carefully watched, lest the heart may not stand well the strain of the sudden assumption of an upright position after the body has so long lain horizontally; and if any cardiac weakness is shown, a heart-tonic should be administered, the best being the sulphate of strychnia, gr. $\frac{1}{30}$ three times a day. Occasionally this drug acts disagreeably upon the unstriped muscles of the intestine, causing griping and infrequent small stools. In such an event digitalis should be given, ten drops of a carefully and freshly made tincture, or one grain of the powdered digitalis, either three times a day. The rest should be kept up for a period of ten or twelve weeks, to the extent, at least, of keeping the patient in bed all but about four or five hours in the day, and during these four or five hours long walks should be absolutely prohibited. Massage, if properly used, will not only prevent the muscles from becoming flabby, but will in many cases strengthen them to such a degree as to decrease the inco-ordination. It should be begun in the second or third week of treatment. It must never be severe, however, and it is always best to watch carefully the person giving massage, to see that this point is observed. How long to administer it is a question that varies from case to case; usually beginning with twenty or thirty minutes of very gentle massage, patients will come to stand forty-five minutes to an hour of it, but in some instances they never can take more than a half-hour without extreme fatigue. The patient should lie quiet for an hour to two hours after each massage. It should be administered every other day for the first week or two, and then daily. Galvanism is an invaluable agent. It should be applied to the spinal column, one pole on the nape of the neck (Fig. 67), the other on the lower dorsal vertebra (Fig. 68), and a current of 3 to 5 milliampères should be passed for three to five minutes every day. In some cases there is great susceptibility at first to the electric current, and it should therefore be begun with great caution, although even in these cases large quantities may be administered in time. In other cases galvanism is not borne well at all, and it is a singular clinical fact, for which I have never been able to find an explanation, that many cases of locomotor ataxia of syphilitic origin cannot take electricity; indeed, I have seen two cases get rapidly worse with very small currents. I am not by any means certain that the so-called crises are not often excited by the administration of electricity. The relief of the pain is sometimes a difficult matter, but usually the best means of combating them is absolute rest. When this does not answer, the following prescription may be written:

℞.—Quin. sulph. . . . . gr. ij.
Sodii salicylat. . . . . gr. j.
Acetanilide . . . . gr. ijss. M.

Ft. in capsul. no. j. Mitte xv. S.—One three times daily, after meals.

Codeia, gr. $\frac{1}{4}$–$\frac{1}{5}$, may be added to this if the acetanilide is not sufficient to overcome the pain, and if the combination depresses the patient at all, strychnia sulph., gr. $\frac{1}{30}$, may also be added. When pains continue in spite of these milder anodynes, 10 drops of the tincture of opium should be given once or twice a day. If, however, the pain is still unrelieved, resort must be had to hypodermics of morph. sulph., $\frac{1}{8}$ grain once or twice a day. It is always advisable, however, to avoid this, because no patient is more prone to form the morphine-habit than one with locomotor-ataxia; indeed, all the above medicines should be sent to the patient, and the prescriptions should not be put into his hands, and if they are marked, "No copy, no repetition," a reliable druggist will not inform him of their contents. Even the tincture of opium can be administered in the form of a tablet triturate. Should the pain still continue, there is often good reason to suspect that the case is syphilitic to a larger degree than had been supposed, and the iodide should be gradually increased until somewhere between 150 and 200 drops of the saturated solution are given in the day. The application of the actual cautery will often very materially relieve the pain, and it should be applied four or five times, but only lightly and superficially. As a further adjuvant to these anodyne measures the faradic brush will be found useful, passing a strong current and brushing it gently over the affected limbs; also warmth applied by means of cloths wrung from very hot water and quickly applied, or even by poultices. Suspension is a new fad that has certainly effected a temporary improvement in all the symptoms of some cases, often to a wonderful degree, as in one case of my own where it was tried as the last resort, and, incredible as it may seem, the patient after two suspensions got out of bed, which he had not left for weeks, and walked down several flights of stairs! Yet he died two weeks after. The suspension-apparatus should be the ordinary one made by all instrument-makers, with head-strap and axillary supports. Suspension should be very gently and gradually done, at first for thirty seconds, afterward, if it has not proved harmful, for one or two minutes every three or four days, and the patient should rest for an hour or two after each suspension. The atrophy of the optic nerve is sometimes seemingly benefited by sulphate of strychnine, gr. $\frac{1}{100}$ to gr. $\frac{1}{30}$, three times a day, and continued for a week or two at a time, then an intermission of several weeks, when it may be resumed again for a week or two. The vesical symptoms should be treated according to the directions in the Chapter on Myelitis (p 229). The vesical symptoms will also be benefited by alkalies, non-nitrogenized diet, and properly selected clothing and underclothing. Galvanism will often be found to be extremely beneficial for the bladder-trouble, and for this purpose the patient should be seated upon a large electrode, covered with a heavy sponge, which should be dripping with hot

water (p. 121). This should be placed under the perineum, and the positive pole should always connect with it. The other electrode should be a large one, and should be placed over the lower dorsal spine. These electrodes being placed in position, a gentle current of 2 to 5 milliampères should be gradually turned on and allowed to pass for five minutes. This electrical application should be made every second day. The so-called crises often resist treatment very obstinately. For the nephritic and laryngeal crises the best treatment is the internal administration of codeia, gr. $\frac{1}{4}$ to $\frac{1}{3}$ t. i. d., or every four hours, or a hypodermic of sulphate of morphine and sulphate of atropine. For the gastric crises, nux vomica, bismuth, papain, codeia, oxalate of cerium, pepsin, and hypodermics of morphine and atropine are the means that are most effective. Moreover, the food should be light and easily digested, and, if necessary, should be peptonized. For the obstinate constipation aloetics, with nux vomica or belladonna, may be given, or cascara sagrada, as recommended on page 229.

PRIMARY LATERAL SCLEROSIS.

Synonyms: Einfache spastische Spinalparalyse (Erb). Tabes dorsal spasmodique (Charcot). Spastische Spinallähmung. Primary lateral sclerosis. Spastic spinal paralysis.

Definition. So-called primary lateral sclerosis is a disease of the spinal cord characterized by contractures, exaggerated motor reflexes, and hasty micturition.

History. Attention was first called to this disease by Erb in 1875, and shortly afterward Charcot wrote upon it Since that time numerous contributions have been made to the subject.

Clinical Symptoms. This is a chronic affection, and the onset is very gradual. The symptoms consist of contractures and exaggerated motor reflexes, the combination of the two classes of symptoms causing a peculiar walk known as the spasmodic or spastic gait. In a pronounced form the contracture is such as to cause abnormal position of the limbs, especially of the flexor muscles, as well as great stiffness, and it can then be readily detected in attempting to flex or extend the affected limb. In less degree, however, the contracture may not be present to the naked eye, although the patient may complain of stiffness in the affected limbs. At this time. however, its presence can often be detected by the following test: Extend the limb fully, tell the patient to make no resistance whatever, and then make a quick, sudden movement of flexion. If there is a beginning contracture, the flexion will be met by a slight muscular click or resistance, which a little experience will easily teach one to differentiate from voluntary resistance (p. 170). The exaggerated reflexes consist of the knee-jerk, sometimes ankle-clonus, jaw-jerk, and hasty micturition. By this latter symptom, to which attention was first called by Seguin in 1873, is meant a desire to micturate that is far less controllable than it should be in a healthy person. In unusual cases the tendon-reflexes of the arm- and hand-muscles may be also

found to be exaggerated. The combination of contracture and exaggerated tendon-reflexes gives rise to the so-called spastic gait. The patient walks upon his toes and the ball of the foot, scarcely touching the heel to the ground, and walks as if upon stiff bed-springs, with a jerky, jiggery motion that is very characteristic. Sometimes, in addition to these peculiarities, the leg is trailed slightly along the ground, the toes shuffling along the floor. It is a matter of habit in my clinic to diagnose this walk almost unfailingly.

Pathology. When the autopsies in so-called cases of lateral sclerosis are examined it is readily seen that the theory that there is a primary affection of the lateral pyramidal columns is scarcely warrantable. The first post-mortem was made by Charcot, and the lesion was found to be a disseminated sclerosis. The second autopsy was made by von Stofella, and although the lesion was found to be in the two lateral columns in about the area of the lateral pyramidal column, the spinal cord was not microscopically examined, and no mention was made of the brain and medulla. Schulz reports five autopsies; in one there was a gliomatous tumor in the medulla extending into the anterior pyramid; in the second a small sarcomatous tumor was found between the pons and the *crus cerebri ad pontem;* in the third case there was a chronic hydrocephalus and no spinal changes whatever; in the fourth there was a pachymeningitis causing myelitis; in the fifth there was a transverse myelitis with secondary postero-lateral sclerosis. Hallopeau's case had a tumor in the medulla. Strümpell describes two autopsies; in the first there was a myelitis of the upper dorsal region; in the second there was a hydromyelus extending throughout the cord. In Friedreich's case there were numerous cerebral extravasations, multiple sclerotic patches in the medulla and spinal cord, with proliferation of the neuroglia, and many patches of softening. Suckling's case was very evidently one of myelitis with subsequent lateral degeneration. John Hopkins, to be sure, reports a case as having only a degeneration in the lateral columns; but he makes no special mention of any examination having been made beyond the upper portion of the dorsal region. The only single case that has been carefully examined and that would seem to be a case of primary lateral sclerosis is one reported by Dreschfeld and Morgan. In this microscopic sections of the brain and the medulla above the lower portion of the anterior pyramid in the medulla are said to have been perfectly normal, and there were no pathological changes observed except in the lateral and anterior pyramidal columns. But one case, out of fourteen reported during a period of nineteen years, is certainly not enough to warrant a pathological assumption, and it is very much more probable that Dreschfeld has overlooked some changes in the upper portion of the pyramidal tract—possibly such slight ones as have been found in some cases of amyotrophic lateral sclerosis in the motor convolutions—rather than that there really is a primary implication of the lateral pyramidal columns.

Duration. The duration in cases of so-called primary lateral

sclerosis will depend to some extent upon the causative lesion, although in every case that I have seen it is eminently a chronic disease, one patient of mine having had it for fifty years, since earliest childhood.

Prognosis. The prognosis of lateral sclerosis is absolutely unfavorable as regards improvement or cure.

Diagnosis. The diagnosis of lateral sclerosis from other diseases is generally easy enough, and the differentiation is to be from the other maladies which are enumerated in "Amyotrophic Lateral Sclerosis," immediately following.

Treatment. The treatment of lateral sclerosis is that of chronic myelitis, except that the exaggerated reflexes can sometimes be materially benefited by the administration of hyoscyamine gr. $\frac{1}{100}$ once or twice a day, or the hydrobromate or hydrochlorate of hyoscine in the same doses. Belladonna also seems to exert a favorable influence upon this symptom in certain individuals, although it fails utterly in others, and the drug should be administered in the form of a reliable fluid-extract, one drop once to three times daily, or the sulphate of atropia, gr. $\frac{1}{100}$ at the same intervals.

AMYOTROPHIC LATERAL SCLEROSIS.

Synonyms: Spastische Spinalparalyse mit Muskelatrophie. Amyotrophische Lateralsclerose. Sclérose latérale amyotrophique (Charcot).

Definition. Amyotrophic lateral sclerosis is a disease of the spinal cord characterized by motor paresis, muscular atrophy, contracture, and exaggerated reflexes, without cerebral, sensory, vesical, or rectal symptoms.

Duration. The average duration of the disease is within three years, but cases have been known to last for ten and even fifteen.

Clinical History. The typical cases usually present three somewhat fairly defined stages, viz.: First, of paresis; second, of atrophy; third, of contracture and exaggerated reflexes. But there are many exceptions to this rule. The onset is generally in the upper extremities, sometimes in one of the fingers. The extension to the lower extremities and to the medulla oblongata is usually made within six to twelve months. Sometimes, however, the onset may be in the medulla oblongata, or in the lower extremities. The paralysis is purely motor. The muscular atrophy is invariably of the whole body of the muscle, and not fibre by fibre, as in progressive muscular atrophy. The tendon-reflexes are exaggerated, especially the knee-jerks, and ankle-clonus is often obtainable; but these symptoms disappear in the later stages. The commingled contracture and atrophy give rise to characteristic deformities, such as the claw-hand, etc., and there is generally a fibrillary tremor of the affected limbs, and sometimes an intentional tremor, *i. e.*, a tremor caused by voluntary movements. The progress of the disease is gradual. The affected muscles usually present some phases of the reaction of degeneration. Death ensues from implication of the vital nuclei in the

medulla oblongata, from general debility, or from pulmonary complications.

Prognosis. The prognosis is hopeless, both as regards life and improvement.

Diagnosis. The disease is to be differentiated from—

Progressive muscular atrophy;
Myelitis of the anterior horn;
Ordinary myelitis;
Postero-lateral sclerosis.

Progressive muscular atrophy begins with a fibrillary atrophy of the muscles, the paralysis is in proportion to this, and there is no contracture or exaggerated reflexes whatsoever; whilst in amyotrophic lateral sclerosis the paralysis is usually a primary symptom, the atrophy succeeds, is of the body of the muscle or muscles, and not fibrillary, and there supervene contracture and exaggerated reflexes.

Myelitis of the anterior horn in the child should never be confounded with amyotrophic lateral sclerosis, for the former is always sudden in onset, is monoplegic in its distribution, and affects only a certain muscular group or groups within that one limb; its paralysis is a flaccid one, and there are no contractures or exaggerated reflexes. Myelitis of the anterior horn in the adult usually begins with motor paralysis of one or more limbs or the whole body, reaching its acme in a few days, muscular atrophy succeeding within a fortnight, and the paralysis remains a flaccid one, contractures and exaggerated reflexes very seldom supervening.

An *ordinary myelitis* in its earlier stage might be confounded with amyotrophic lateral sclerosis, but the progress of the former affection should render a mistake impossible, as ordinary myelitis has pronounced sensory, rectal, and vesical symptoms, which are never present in amyotrophic lateral sclerosis.

Postero-lateral sclerosis, or combined sclerosis—*i. e.*, that form of combined myelitis in which the posterior and lateral columns are simultaneously affected—would have superadded to the lateral sclerosis the symptoms of locomotor ataxia.

Pathology. The lesion of amyotrophic lateral sclerosis always consists of a degeneration in the lateral and anterior pyramidal columns and of destruction of ganglion-cells in the anterior cornua of the spinal cord and the motor nuclei of the medulla oblongata. The implication of the lateral columns is always more marked than that of the anterior pyramidal columns, and sometimes the latter are scarcely affected at all. The lesions of the ganglion-cells are the same as are seen in poliomyelitis anterior, and consist of morphological alterations in their structure. Some of the cells disappear entirely, some lose their processes, some become smaller in size, some are highly pigmented. It has been a belief with many authors that the cellular alterations in these anterior cornua are due to precedent arterial alterations, but there has been no proof advanced of this view. The lesions in the pyramidal columns consist of impairment or destruction of the nerve-tubules, and proliferation of the connective

tissue, with the occasional presence of fatty granules and *corpora amylacea.* Inasmuch as the lesions in the pyramidal columns and in the anterior horn or the bulbar nuclei are always bilateral, there has been a suspicion in the neurological mind that the affection was of the nature of a secondary degeneration of the pyramidal columns extending into the anterior horns. In some cases there has been found to be a marked degeneration of the fibres running from the motor convolutions, whilst in the other cases the degeneration has been traced up into the pons, the crura cerebri, and the internal capsule, and in still other cases the degeneration has not been observed above the decussation. In none has there been grave organic disease of the upper part of the pyramidal tract. Whether, therefore, the cases reported as being without lesion above the decussation were so minutely examined as to detect the slight alterations which others have found, is a question that very naturally arises. At the present time we cannot, for this reason, distinctly affirm or deny that the lesion in the pyramidal columns is a secondary lesion, although the probabilities are strongly in favor of its being so. In many cases of poliomyelitis anterior the lateral pyramidal column has been found secondarily affected; in many cases of lesion in the cerebrum the anterior horns have been found diseased, although the pyramidal tracts were intact. In several cases of disease in the cerebrum muscular atrophy has supervened without intermediate lesion of the pyramidal tract or the anterior horn, or even without lesion of the peripheral nerve; from which facts it is easily to be seen that there is a close connection pathologically between the motor convolutions, the pyramidal tract, the anterior horn, the motor nerves, and the muscles.

Treatment. As will be very evident, the treatment of amyotrophic lateral sclerosis will in some cases depend upon the causative lesion, if that can be diagnosed. In most cases, however, this is not possible, and the treatment must be experimentally that of chronic myelitis.

THE COMBINED SCLEROSES OF THE CORD.

Synonyms: Progressive spastic ataxia. Combined fascicular scleroses. Ataxic paraplegia.

Not infrequently cases are observed with the combined symptoms of locomotor ataxia of a mild type and spastic symptoms, with motor paresis; and, in 1885, Ormerod analyzed twenty such cases, whilst, in 1886, Grasset collected thirty-three more, and, in 1887, Dana based a paper upon forty-five. Since then numerous cases have been reported by different authors.

The lesions are generally in the lateral or anterior pyramidal tracts, or both, the cerebellar tracts, the lateral column proper, and the columns of Goll. (Fig. 1.) The dorsal region is usually most affected. The columns of Goll, the lateral pyramidal tracts, and the cerebellar columns are often affected throughout their whole course. The columns of Burdach are sometimes encroached upon in the lower or upper parts of the cord. When the lateral columns are implicated,

the disease sometimes involves the column of Gowers, and, inwardly, the lateral mixed column. The long systems of fibres, such as the column of Goll, the pyramidal tract, and the cerebellar column, are generally affected throughout most of their length; and the degeneration usually extends, contrary to the Wallerian law, in the opposite direction to the course of the normal nerve-sensations or impulses; so that it runs downward in the columns of Goll and the cerebellar tracts and upward in the pyramidal tracts, being less in the lower levels of the first-named columns and in the upper regions of the latter ones. In some cases the cells of the anterior horns of the cord are implicated, and also the cells of the posterior horns and of the column of Clark. There is, in rare cases, a slight and chronic meningitis of the pia mater. The lesions are generally superficial, and do not affect the vital functions of the cord to anything like the extent that is seen in ordinary transverse myelitis or Landry's paralysis. In some of the cases there is a commingling of diffuse lesions and degeneration of the tracts that have been named; and this is more especially apt to occur after an ordinary myelitis, giving rise to secondary degenerations. The combined scleroses are observed in conjunction with many affections of the cerebro-spinal nervous system, such as general paresis, chronic mania, Friedreich's disease, locomotor ataxia, myelitis, and so-called spastic paraplegia. It is a question whether the disease is a primary one or is secondary—*i. e.*, whether the degeneration of the different columns is secondary to some pre-existing lesion of the brain or cord, or whether they start primarily and simultaneously in these different columns. It is very certain, as we have seen, that such general lesions as general paresis, chronic mania, and myelitis may have combined scleroses associated with them, and it therefore seems most probable that the general lesions are the cause and the starting-point of the degeneration in the different columns Whether any cases are primary is a matter that is, to my mind, very questionable, for a primary disease of the long system-fibres of the cord has never yet been demonstrated, and, in the case of so-called lateral scleroses and locomotor ataxia, the early doctrine that the degenerations observed in the different columns were primary has been gradually disproved. (See Chap. "Lateral Sclerosis.")

As might be supposed in advance, the symptoms of the combined sclerosis are commingled symptoms of locomotor ataxia and lateral sclerosis, except that the clinical picture is a milder one than in individual cases of either one of these two diseases. The disease is chronic, and the onset gradual. There is first noticed a motor weakness of the lower extremities, usually more pronounced upon one side. Then the patient begins to show some inco-ordination of movement, such as being unable to walk steadily in the dark, swaying with the eyes closed and the feet approximated, the walk becoming unsteady, sometimes tottering. If at this time the muscular strength is tested, there will be found to be muscular weakness, contrary to what is observed in locomotor ataxia. This ataxia may increase very greatly in degree. The sensory and reflex symptoms of locomotor

ataxia, however, are generally lacking. There is seldom any of the lightning- and stabbing-pains or much impairment of sensation, and the tendon-reflexes of the knee and foot, instead of being lost, as is generally the case in locomotor ataxia, are exaggerated—often greatly, so that the knee-jerk is spastic, and the foot-clonus may be readily evoked. Gowers believes that a dull sacral pain is quite frequent, and sometimes a similar dull pain is felt in the legs or in the spine after fatigue, and is an early symptom. The cincture sensation, or feeling of a belt about the waist, so common in ordinary myelitis, is almost invariably lacking. The upper extremities are usually normal, but they may present symptoms similar to those of the legs. There is seldom any muscular atrophy. The sexual power is often impaired at an early period, and the bladder and rectum are frequently implicated. There is sometimes a loss of the pupillary reflex to light, and occasionally also to accommodation. Optic-nerve atrophy is far less frequent than in locomotor ataxia. The ocular muscles are sometimes affected. There is no nystagmus. In some cases slight impairment of articulation has been noticed, and slight tremulous movements of the facial muscles without mental change. As the disease progresses the motor weakness and the spastic symptoms increase, and the case comes to present the picture of a genuinely ataxic paraplegia, which is the apt name that has been given to it by Gowers. Complete paralysis may result. The malady, as has been said, is a chronic one, lasting many years, and death occurs from renal disease and the trophic lesions incidental to all forms of myelitis.

The diagnosis of the combined scleroses must be from locomotor ataxia, lateral sclerosis, myelitis, and lesion of the cerebellum or its peduncles. This diagnosis can usually be made with ease. From locomotor ataxia it is to be distinguished by the exaggerated knee-jerk and foot-clonus, the former of which is infrequent and the latter unknown in locomotor ataxia, as well as by the absence of the typical stabbing- and lightning-pains of the latter malady. From lateral sclerosis it is to be distinguished by the marked element of ataxia that is observed in the walk, in the swaying with the eyes closed and the feet approximated, and, when the upper extremities are affected, in the inability to perform finely co-ordinated movements with the arms and hands. From ordinary myelitis it is to be differentiated by the exceedingly gradual onset, by the lack of marked sensory symptoms, severe paralysis of the bladder and rectum, and the cincture sensation; but it should be remembered that the disease may follow upon a myelitis, in which case the history will make the matter clear. From lesion of the cerebellum and its peduncles it is easily distinguished, because the ataxia of cerebellar disease is a peculiar swaying and staggering, like that of a person slightly intoxicated—so-called titubation; and there will be present symptoms of an intracranial lesion, such as headache and paroxysms of vomiting, whilst in some lesions of the cerebellar peduncles there are semi-rotary or markedly staggering movements to one side.

The prognosis of the combined sclerosis, so far as we now know, is grave as regards the cure, and most cases continue for many

years. one of Strümpell's lasting thirty, whilst, on the other hand, some have terminated fatally in a few years. The disease, however, is probably, as has been said, but a mixture of symptoms, and the future will probably separate it into several maladies, so that the prognosis may undergo much modification. I am myself inclined to think, as I have already said, that many of these cases are really cases of spinal syphilis.

The treatment of the disease should be upon the same principles that have been laid down when treating of locomotor ataxia and myelitis. The systematic and careful use of the faradic current of high tension, as well as the static current, are spoken of favorably. In all cases the patient should be treated thoroughly for syphilis by means of the iodides and mercury, as indicated in Chapter VIII., "Syphilis of the Nervous System."

SYRINGOMYELITIS.

Of late years there has grown up a voluminous literature upon the symptomatology and the pathology of syringomyelia, which, as the name denotes, consists of certain peculiar cavities in the substance of the spinal cord. This pathological condition has been recognized for fully two hundred years past, and to it Ollivier proposed the name above. There are in reality two cavities of this kind within the spinal cord: one termed hydromyelus, consisting of a dilatation of the central canal lined by cylindrical epithelium; and the true syringomyelitis, due either to an infiltration of the spinal parenchyma with gliomatous cells, or to a peculiar connective-tissue formation known as gliosis. Either may be congenital or acquired. Besides these there may be mixed forms, consisting of hydromyelus and syringomyelitis—*i. e.*, congenital and acquired hydromyelus with congenital and acquired syringomyelitis; or there may be a hydrosyringomyelitis—that is, there may be a hydromyelus or dilatation of the central canal continuous with a cavity or syringomyelitis extending out into the cord, such as Schaffer and Priesz have recently described. Hydromyelus is, as has already been said, a dilatation of the central canal of the cord. This, however, is not always in the centre in the site of the fully developed central canal, because the embryonic central canal is represented often by a cavity extending into what is in later life the posterior fissure, which is generally obliterated in the process of development, but which may remain in adult life separated from the canal in the centre. In such a latter event, however, this cavity possesses an epithelial lining, as has just been stated. Syringomyelitis is a cavity entirely outside the site of the adult or embryonic central canal, never possesses an epithelial lining, and its site varies in each case according to the molecular causes leading to its formation, bearing no constant relation to the adult or embryonic central canal. There has been much discussion as to the mode of formation of the cavity of syringomyelitis. Some have held that the anterior portions of the posterior columns have a peculiar predisposition to pathological alterations, so that in this area

new formations readily occur in which the centre breaks down into a softened mass and is absorbed, leading to the formation of the cavity. Others have maintained that hydromyclus and syringomyelitis are identical—*i. e.*, that the syringomyelitis found in adults is the remains of a congenital hydromyelus. This old view of Leyden's, however, has been abundantly disproved. Others have asserted that both hydromyelus and syringomyelitis are caused by the pressure of tumors, especially gliomata, inducing venous stasis and œdema. But that tumors are not necessary to the formation of such cavities is proved by the case of Joffroy and Achard, who in their turn maintain that there is a non-gliomatous form of syringomyelitis; and this view has been supported by two cases of Schaffer and Priesz. As has already been mentioned, either hydromyelus or syringomyelia can be congenital or acquired; that they are identical, however, is disproved by the histological differences which have been already alluded to. That the two conditions may be combined has been thoroughly proven, especially by the fifth case of Schaffer and Priesz, where a hydromyelus of the cervical enlargement was found in the same cord with a glioma of the dorsal region, the two becoming confluent at a certain level. In the cases of syringomyelitis due to glioma the pathological alterations usually begin with an infiltration of small cells, consisting of connective-tissue nuclei or embryonic cells, and at the same time there is a production of exceedingly fine neuroglia, so that the normal elements of the cord are displaced. In the centre of this gliomatous mass there is a tendency to disintegration and a cavity results. These cavities are usually in the central gray matter about the commissure, usually extending backward into the posterior gray horns or posterior columns, and sometimes extending forward into the anterior horns on one or even both sides. The extent of the cavity upward and downward in the cord varies, so that it may only be found for a short distance, or it may extend through the whole cord. The lower cervical and the upper dorsal regions are especially prone to be affected. The wall of the cavity consists of a dense connective tissue extending for some distance beyond its margins; the cavity itself is filled with fluid of a serous or hyaline consistency. In the non-gliomatous cases described by Joffroy and Achard no tumor of any kind was apparent, but there was around the cavity a sort of amorphous exudate surrounding the vessel, often infiltrating the whole vascular tract. The neuroglia of the white and gray substance was displaced and disintegrated by this exudate. This neurogliar wall was formed by greatly condensed fibrillary neuroglia, with all the characteristics of an old neurogliar sclerosis which had reached a strong degree of intensity, almost like that of a cicatrix, and which the observers considered to be of secondary formation, as they could perceive in it none of the characteristics of young neuroglia or of neoplastic tissue, such as is seen in the periphery of tumors. At one point the process of disintegration of the nervous tissue by the amorphous exudate was plainly observable. In this case of non-gliomatous syringomyelia there was noticed an enormous varicose dilatation of the veins of the pia mater

(which has also been observed in the gliomatous cases), as well as abundant leucocytes in the perivascular sheaths, which leucocytes were also observed in the interior of the vessels pressed against the internal wall, as if the circulation had been retarded. The cause of the stasis of the circulation is as yet a mystery, although it was at first supposed that it was due to the pressure of the tumor; but this case of Joffroy and Achard shows that it was found in cases where there is no tumor. Rosenbach and Schtscherbak have produced such stasis experimentally by compression of the spinal cord, and have also observed similar exudates. Joffroy and Achard think that in their case the relaxation of the venous circulation was produced by arterial thrombosis, as they had found several capillaries affected with obliterating arteritis. These same authors call attention to the fact that many cases of syringomyelitis that have been reported as caused by glioma are really of a nature similar to their own, and they maintain that all the scleroses of the spinal cord are of a similar nature, and should be denominated by the generic name of *gliosis*, meaning by this the peculiar histological formation described by Chaslin (*vide* Chap. IX., "Epilepsy"). Weigert agrees with them to the extent of believing that the presence of a mass of neuroglia around a syringomyelitic cavity does not necessarily signify glioma, but may be a secondary formation. Indeed, Joffroy and Achard go still further in maintaining that the presence of a compact and circumscribed mass of neurogliar proliferation is not necessarily glioma, because in such cases the structure of the so-called tumor is not identical with the neoplasms to which this name has been given; and Weigert also declares that in cerebral gliomata the neurogliar fibrillæ are almost wanting or are much less abundant than in gray degeneration or syringomyelia, whilst in this particular gliosis which we are dealing with there are certain characteristics distinguishing them from the gliomatous tumors, such as their development longitudinally, strand-like, as well as their central softening, with the production of a cavity surrounded by a sclerotic and cicatricial-like wall. Francotte has observed a case of syringomyelitis in conjunction with an acute myelitis, and Kiewlicz has reported another occurring with a transverse myelitis and multiple sclerosis. Charcot has also seen this affection with hypertrophic cervical meningitis, and Köller, Vulpian, and Simon have also observed it in conjunction with chronic meningitis at the points where the cavity began, whilst Oppenheim has found it also with meningomyelitis of traumatic origin, and Silcock with meningeal adhesions of a general spinal sclerosis.

The disease is an eminently chronic one, slowly progressive, some cases lasting even twenty years, but there are remissions.

I have been able to count up 167 autopsies. Upon the symptoms observed in these various cases there have been vigorous attempts made to offer a symptomatology of the disease sufficiently precise to make a diagnosis, and it is now quite a fashion in this country to report so-called cases of syringomyelitis. Very few *ante-mortem* diagnoses have been made, however, although recently Dercum has

reported a brilliant one. It has already been stated that the cavity affects by preference the lower cervical and the upper dorsal region of the cord, and by reference to pp. 81 and 82 it will be seen what groups of muscles are therefore most likely to be affected. In this lower cervical and upper dorsal region the centre of the gray matter is especially prone to be involved, and the cavity extends out into the posterior cornua, sometimes into the lateral columns. *A priori*, therefore, we should expect that the symptoms of such a cavity would be those of sensory impairment, vasomotor and trophic symptoms, muscular atrophy, occasionally contracture; and as the cavity is, as a rule, predominantly unilateral, certain of these symptoms would prevail more upon one side. As a matter of fact, great stress has been laid upon the sensory, vasomotor, and trophic symptoms. The impairment of sensation, it is claimed, consists of what is termed disassociated anæsthesia; *i. e.*, marked impairment of the senses of temperature and pain, whilst the senses of tact and muscular sense are either altogether unimpaired or very slightly affected. The vasomotor and trophic disturbances consist of cyanosis, coldness, bullous eruptions, ulceration, abscesses, even atrophy and fragility of the bony structures, and a diminution of perspiration. It is, therefore, asserted that we may make a diagnosis of syringomyelitis in any case of chronic spinal disease in which there are areas of disassociated anæsthesia, marked vasomotor symptoms of the kind that have been described, muscular atrophy of certain groups of muscles, ataxia, and contracture; these symptoms being predominantly unilateral and varying greatly in their degree in different areas. The diagnosis is a difficult one to make, in proof of which is the remarkable fact that Schultze, who has been the most enthusiastic advocate of the possibilities of such a diagnosis, read his first paper upon the subject in 1878, and yet, up to the present time, seventeen years afterward, he has never made a single one before death. In especial is it difficult to make a differential diagnosis from Morvan's disease (see "Morvan's Disease"), in which the autopsies so far made have shown that the lesion is syringomyelia, although it is not yet determined whether this same symptom-group may not have other pathological lesions causative of it, as Morvan himself stoutly believes; whilst Hanot has reported a case of Morvan's disease subsequent to typhoid fever in which no autopsy was made, it is true, but yet in which the symptoms followed the typhoid fever at too quick an interval to permit of the supposition of a syringomyelitis.

If the symptomatology, as it has been described, should prove with time to be accurate, the diagnosis ought readily to be made from such other diseases as—

Amyotrophic lateral sclerosis;
Progressive muscular atrophy;
Transverse myelitis;
Disseminated sclerosis;
Locomotor ataxia;
Cervical hypertrophic pachymeningitis;

Alcoholic paralysis;
Hysteria;
Anæsthetic leprosy.

Amyotrophic lateral sclerosis and *progressive muscular atrophy* are bilateral diseases, and have no sensory disturbances whatever, and no trophic disturbances except the muscular atrophy of the progressive muscular atrophy.

Transverse myelitis is also a bilateral disease, frequently of acute origin, in which the marked trophic disturbances causing necrosis of bone, abscess, and whitlows are not observed.

In *disseminated sclerosis* the symptoms are almost invariably bilateral, and there is a degree of tremor which is not usually observed in syringomyelitis, whilst the trophic symptoms are not so great as in the latter lesion.

Locomotor ataxia, too, is a bilateral disease, and the disassociated anæsthesia has not been observed in it so far as we now know, whilst the trophic disturbances are never of the extent of those which are claimed for syringomyelitis.

In *cervical hypertrophic pachymeningitis* the great pain in the cervical region radiating down into the upper extremities is greater than is usually observed in cases of syringomyelitis, although occasionally it may be difficult to make a differentiation from this symptom; but in this form of meningitis the trophic disturbances are never of the extent of those observed in syringomyelitis.

In certain cases of *alcoholic paralysis* and *hysteria* the disassociated anæsthesia has been observed, but in alcoholism the knowledge of the causation and the other symptoms, and in hysteria the general symptoms of hysteria itself, will enable us to make the diagnosis, although it must be remembered that in syringomyelitis, as well as in hysteria, there has been observed a concentration of the field of vision.

In *anæsthesic leprosy* there is, according to Leloir, a prodromic period, or a period of invasion; an eruptive period, which may be short or excessively prolonged, and which is characterized by a cutaneous eruption or by an eruption along the nerve-trunks, or, rarely, the cutaneous eruption may be lacking; third, a stage of nervous implication. The prodromic period has such general symptoms as fever, chills, tendency to slumber, painful digestion, rheumatoid and neuralgic symptoms, etc., of such marked intensity that the patient is usually confined to bed. The eruptive period is characterized by macula, erythema, or pigmentary macula. In the stage of nervous involvement there may be well-marked anæsthesia, usually not, however, so far as we yet know, of the disassociated form, and the trophic disturbances may be as marked as in Morvan's disease. The diagnosis, however, can be made by attention to the precedent stages.

MORVAN'S DISEASE.

In 1883 Morvan described a disease which he denominated analgesic paresis with whitlow of the superior extremities, or pareso-

analgesia of the superior extremities, but to which, in the twelve years that have since elapsed, the name has come to be given of Morvan's disease. He states that he had been observing these cases for upward of thirty years, and his attention was first called to the matter by having a patient come to him who had a whitlow for which he recommended incision, and who, much to his surprise, manifested no evidence of pain whatever when the incision was made. Morvan has since made a number of communications upon the same subject. The malady affects the upper extremities, being at first localized in one of them, usually in the forearm and hand. The first symptoms are those of neuralgia, the pain being often very severe, and these are followed by paresis and analgesia, finally by one or more whitlows, with necrosis of the phalanges. The disease proceeds in stages. When the neuralgic period has lasted for several years, perhaps, the paralytic, analgesic, and trophic symptoms appear, and extend into the other upper extremity. Atrophy of the muscles paralyzed is generally observed, or there may be analgesic segments of the limb. In 1886 Morvan had reported nineteen cases, four of which only were in males. Youth is a predisposing age, as in twelve of these cases the age was between twenty and thirty years, and the others beyond this. In six there was seemingly a traumatic origin. Morvan had also seen, contrary to his first experience, the malady affect the lower extremities, although he had never observed that the muscles of the trunk were affected. The analgesia had sometimes involved the whole upper limb, in three cases the corresponding side of the neck, the face, and the scalp to the vertex, and in two cases the trunk as far as the last dorsal pair. Hyperidrosis had also been observed. In no case had paresis been witnessed without analgesia, but in three cases there was analgesia without paresis. In most of these cases disassociated anæsthesia has been observed, but in some of them there has been an impairment of all the four senses of touch, pain, temperature, and muscular sense.

The only autopsies that have yet been made are the three by Joffroy and Achard, and in every one of them a syringomyelitis has been found. Hanot, however, has reported a case as occurring shortly after a typhoid fever—years too soon, it would seem, to permit of the supposition of a syringomyelitis being present. The differential diagnosis has been dealt with fully in "Syringomyelitis," and it is yet a question whether Morvan's disease is a form of syringomyelitis, or whether it may not be due to other causes.

TUMORS OF THE SPINAL CORD.

Tumors of the cord are most frequently syphilitic or tuberculous, although parasitic tumors occasionally occur, and in some rare cases spinal neoplasms are congenital. The tumors may be outside the dura, inside this membrane, or in the substance of the cord. The extra-dural forms are lipoma, echinococci, enchondroma, sarcoma, cancer, and connective-tissue proliferations from osseous disease. The intra-dural neoplasms are chiefly syphilomata, sarcomata, myxomata,

tubercular, parasitic growths, echinococci, cysticerci, and fatty growths, although the last five are rare. In the parenchyma of the cord syphiloma, glioma, sarcoma, myxoma, and tubercular tumors occur, the first two being the most frequent, and cysticerci are occasionally met with.

The symptoms of spinal tumors should be divided into two classes: general and localizing ones. The general symptoms are pain and malaise. The pain is often severe, and varies much in character, being darting, stabbing, or dull and aching. Its intensity is usually great. The malaise is generally in proportion to the pain, although in cancers it may be disproportionate. The localizing symptoms are those caused by implication of the cord at different levels, and of these the best idea can be gained by a study of the table on page 82. However much the localizing symptoms may vary in degree, they are invariably accompanied by motor and sensory paralysis and by muscular spasm.

The diagnosis of spinal tumors is a difficult one, and they must be distinguished from such affections as myelitis, locomotor ataxia, spinal syphilis, spinal tuberculosis, neuritis, caries of the vertebral column, Pott's disease, syringomyelitis, and traumata of the cord.

When myelitis is of the type of poliomyelitis anterior the affection occurs in a child usually, and is purely motor in its symptoms, consisting of a paralysis of certain muscles in a limb, generally the arm- or leg-type, with atrophy and reaction of degeneration, and there are no vesical, rectal, sensory, or spastic symptoms. When the myelitis suspected is a transverse myelitis, the symptoms are generally subacute, reaching their acme in a few days or a week, and not attended by the pain that is characteristic of spinal tumors.

Locomotor ataxia has only one symptom giving it a resemblance to spinal tumor, and that is the pain; but in locomotor ataxia, although the pain may be darting, stabbing, and severe, it varies in location, is vagabond in character, and is also paroxysmal and not continuous as in spinal tumor; nor are there the motor paralysis and spastic symptoms which are present in spinal tumor. Besides this, locomotor ataxia has an optic-nerve atrophy associated with it in a certain proportion of cases, which is not usually seen in cases of spinal tumor.

Spinal syphilis, it should be borne in mind, may cause a tumor, and this is generally associated with symptoms of diffuse spinal or cerebro-spinal syphilis. In cases of spinal syphilis I believe that there is a great tendency to exaggerated tendon-reflexes, particularly of the quadriceps extensor tendon (knee-jerk), and that the paralysis is either motor or mainly motor, whilst pain is an infrequent symptom; and in cerebro-spinal syphilis there will also be symptoms of cerebral lesion which are entirely lacking in cases of spinal tumor. But if a spinal tumor should be syphilitic, and the symptoms of general spinal implication by the syphilis should be subsidiary, the diagnosis may become very difficult, and it can only be made tentatively upon a knowledge of the syphilitic infection, conjoined with the results of mercurial or iodide treatment.

Spinal tuberculosis may give rise to a tumor alone, or to diffuse tuberculous infection of the cord, associated with the formation of a neoplasm. In the latter case the history of the tuberculous heredity or infection, with a knowledge of precedent pulmonary implication, would aid us greatly in making the diagnosis. If, however, there should be no positive history of tuberculous infection, and no evidence of general implication of the spinal cord by tuberculosis, the diagnosis may be made by exclusion; in other words, syphilis, caries, and Pott's disease must be excluded, when the diagnosis would lie between a tuberculous and some other form of tumor, and the differentiation would be impossible.

The diagnosis from neuritis should not be difficult, because in neuritis the affection would be limited to a single nerve-trunk, or in multiple neuritis there would be an acute or subacute onset of the affection, and besides this, the pain of neuritis is not apt to be so sharp and lancinating as in spinal tumor.

In caries of the vertebral column there will be a greater immobility of the vertebral column than in cases of spinal tumor, and there will not be the pain. The diagnosis of spinal tumor is often extremely difficult, however.

In Pott's disease the diagnosis can usually be made with ease by means of the characteristic vertebral deformities, and the history of rhachitis or tuberculosis as the predisposing cause.

The diagnosis between spinal tumor and syringo-myelitis (which is often but a form of tumor) can be made by reference to the details given in the Chapter on "Syringomyelitis."

Trauma of the cord can be readily diagnosed by a history of the trauma, with probably the consequent vertebral deformity.

The prognosis of spinal tumor is unfavorable, except in the syphilitic cases which have not caused irreparable structural mischief, and whether this has been done can only be determined by syphilitic treatment.

The treatment of spinal tumors should be by means of the iodide and mercurials as has been recommended in Chapter VIII., "Syphilis of the Nervous System." If these should prove to have no effect, an operation upon the spinal cord holds out some chance of relief.

TRAUMATA OF THE CORD.

Traumata of the cord consist of injuries to the cord itself or to the vertebral column. When the cord itself has been injured, hemorrhage may occur, either extra- or intra-dural, in the pia mater, in the substance of the cord itself, or the substance of the cord may be lacerated, and this laceration may give rise to myelitis with softening. Some authors have stated that the cord cannot be injured unless the vertebral column has sustained a fracture, but this statement, which is of great importance in a medico-legal aspect, is incorrect, for a number of cases have been reported (and I have had several myself) in which myelitis has occurred from traumatism without the slightest evidence of fracture of the vertebra. This so-called concussion of

the cord gives rise to myelitis, and not to the symptoms of so-called railway spine, for these latter of late years have been shown to be really traumatic neurasthenia. (Chapter XXII., "Railway Injuries, etc.") The symptoms of injury to the cord, either direct or from fracture of the vertebræ, are those of myelitis, and these symptoms will vary in character according to the level of the cord that is affected, and what the symptoms are can be best realized by a study of the table on p. 82. It must also be remembered that this is of very great medico-legal importance. The symptoms of traumatic myelitis may not occur immediately after the injury, but they may come on gradually, although in all cases there should be a distinct chronological sequence. There is one symptom, however, that is very frequent in traumata of the cord, and that is pain, which may continue for years—in one case of mine extending over twenty years, although a very fair recovery was made. This pain may vary in site according to the portion of the cord that is injured, and in the course of time it usually passes into a neuralgia, which is excited by changes in the weather, such as the approach of storms, the passage of storms, or the period of clear weather (the so-called *anti-cyclone*) succeeding storms, or by fatigue or failure of general health; and it often occurs in paroxysms which have no ascertainable cause.

The diagnosis of injury to the spinal cord or the vertebral column is usually made with ease. In the first place, there is the history of the trauma, and then a careful examination will discover some abnormality in the position of the vertebræ, or some symptoms indicating disease of the spinal parenchyma. As these cases so frequently have a vivid medico-legal interest and occupy so much of the attention of our courts, every case should be examined with the most minute care. The patient should be stripped, made to bend his body in every direction, and the line of the vertebral column should be carefully traced, so as to test the mobility of the vertebral column. Then sensation should be minutely examined, testing the sense of touch, temperature, pain, and muscular sense (*vide* Chapter II., "Tests of General Motor and Sensory Symptoms"). The condition of the bladder should be carefully inquired into, and also that of the rectum. The patient having been stripped, as has been advised, the muscular groups should be carefully examined for losses of contour which may indicate atrophy. The skin of the affected parts should be carefully observed so as to ascertain whether it is unusually glossy, whether the fingers or toes are bulbous, or whether the surface-temperature is decreased. The power of motion should also be carefully tested by ascertaining the strength of the various muscular groups. The tendon-reflexes, especially the knee-jerk, should be interrogated. When such a careful and minute examination as this is made by a competent observer, no possible symptoms should escape. In making this prolonged series of tests, such diseases as traumatic neurasthenia, hypochondria, and Pott's disease should be excluded, and the possibility of the patient being a malingerer should be constantly remembered. In regard to malingering, however, I desire to impress strenuously one fact,

namely, that it should not be assumed, as is too generally done, especially by surgeons in the employ of corporations, that the patient is shamming. The importance of a fair and impartial attitude in this matter can only be appreciated by one who has had large experience in accident-suits. The diagnosis from traumatic neurasthenia is made readily enough, for in the latter the symptoms are general and often commingled with an element of hysteria, whilst in localized spinal trauma the symptoms are local, and there is seldom any hysterical element. In traumatic neurasthenia, also, there is very frequently a concentration of the field of vision and a degree of localized, symmetrical, or bilateral anæsthesia, occupying a segment of limb, and these two symptoms are not observed in the localized traumatic spinal affection. In a large number of cases of traumatic neurasthenia, too, there is no paralysis of motion, although there may be of sensation, but this never occurs with traumatic myelitis. The diagnosis of hypochondria can be readily made by anybody who is expert enough in neurological matters to know that no true symptoms of myelitis are present, and that the patient has the mental characteristics of the hypochondriac. The malingerer can be readily detected by the competent neurologist who is adept enough to penetrate the sham presentation of a myelitis, many symptoms of which cannot be simulated, such as exaggerated and spastic knee-jerk and foot-clonus, loss of pain-sense, muscular atrophy, the reaction of degeneration, and the contractures. Paralysis of the rectum may be claimed, and it may be difficult, for obvious reasons, to ascertain whether the patient is telling the truth or not. To a certain extent this is also true of asserted paralysis of the bladder, but it is sometimes possible to test this statement by one of two methods. In the first place, it may be casually inquired of the patient as to how long he can retain the urine. If he states that he can do this for only a short time, it may be possible to engage him in conversation, or to keep him upon the witness-stand for a period long exceeding that which he has mentioned. If, on the other hand, he asserts that he cannot pass the urine, ask him how long it has been since he last urinated, and if he says that it has been several hours before, the insertion of a catheter and the determination of the amount of retained urine will test the truth of his remark. Unfortunately, however, the last of these methods cannot be often employed nowadays, in the State of New York at least, since the courts have ruled that defendants in damage-suits have no right to a medical examination of the plaintiff. In the second place, an examination of the urine may be of value. If the urine is withdrawn by catheter, and it presents evidence of a cystitis, this would be proof that the patient's statement of vesical paralysis was probably true. Let the reader bear carefully in mind, however, that the converse of this proposition is not good legal proof, for it sometimes happens that a patient has vesical paralysis, causing retention or incontinence of urine, without cystitis. This test, therefore, has only a positive value, and a very limited negative one.

The treatment of traumatic myelitis should be the treatment of an ordinary myelitis, unless there is such evident fracture of the verte-

bral column as to make it evident that there is compression, when a spinal operation should be done. Care must be taken, however, not to operate too soon unless there is such probable compression, for I have seen a number of cases of traumatic myelitis recover without any surgical procedures. If it should be thought advisable to resort to a surgical operation, however, this should be done as promptly as it has been decided upon; for whilst, as I have stated, I have seen cases recover without operation, I have yet seen other cases of indubitable compression left so long that irreparable mischief had been done to the spinal parenchyma, which could have been entirely or largely prevented by an early operation. As regards surgical operation, therefore, cases of traumatic spinal lesion should be divided into three classes: those in which there is indubitable evidence of compression, those in which there is no evidence of compression, and those in which the evidence of compression is doubtful. The first class should be promptly operated upon; in the second class of cases an operation is useless; the third class should be put under careful treatment for myelitis until such time as they have ceased to improve, and, when this cessation of improvement has continued for a period of two or three months, an exploratory operation should be done.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

Synonyms: Bulbar paralysis. Progressive bulbar paralysis. Progressive Bulbärparalyse. Paralysie musculaire progressive de la langue, du voile du palais, et des lèvres.[1] Atrophie primitive des noyaux moteurs.

Definition. A motor paralysis, with muscular atrophy, affecting the motor cranial nerves and attached muscles, especially the hypoglossal, and certain filaments of the facial and the spinal accessory.

History. As far back as 1825 Robinson gave an incomplete description of this disease, in a letter to Sir Charles Bell. But Duchenne led the way in this, as in so many other spinal diseases, by the first recognition of it as a clinical entity in 1861, narrating thirteen cases. Baerwinkel (1861), Schultz (1864), and Wachsmuth (1864) located the lesion, upon theoretical grounds, in the medulla oblongata. But the first autopsies, two in number, were made by Charcot and Joffroy in 1869, and Leyden made two more in 1870. Since then a number of microscopical examinations have been made by Maier, Charcot and Gombault, Duval, Raymond, Pierret, Troisier, Hayem, Duchenne, Joffroy, and Birdsall.

Symptoms. The prodromata of this disease are either absolutely wanting, or are very slight. When they are present, they may consist of vague pain in the neck and back of the head, slight vertigo, feeling of contraction about the chest and breast, and occasional dyspnœa. Krishaber has noticed a loss of the reflex excitability in

[1] This was the name by which it was first described by Duchenne.

the pharynx, larynx, trachea, and œsophagus, although the tactile sensibility was maintained perfectly. The symptoms are those appertaining to a paralysis and atrophy of the tongue, lips, the lower part of the face, palate, and finally the larynx and heart. It has been a mooted question among the older authors whether the paralysis or the atrophy was primary, but, although this is a matter of subsidiary importance, the atrophy is undoubtedly the first symptom in the majority of cases.

The lingual affection generally first attracts the patient's attention because of some slight difficulty of speech. Upon examination of the tongue, it may then be found atrophied, and, if this has proceeded to a certain degree, the organ may have a fissured and furrowed appearance, or may display minute losses of muscular tissue on the edges, causing these to be notched or crenulated. Great atrophy of the tongue is not only visible to the eye by the diminished size of the organ, but gentle palpation between the fingers will show that the muscular tissue has decreased, and that what remains is much softer than normal. The tongue is often agitated by minute fibrillary tremor. Its usual movements are made with difficulty, so that pronunciation is impeded, morsels of food cannot be swept around the buccal cavity with the usual certainty, and deglutition may be interfered with. It must be remembered, however, that this lingual difficulty of deglutition is something altogether different from that which may occur when the muscles of mastication are affected. In pronun-

Fig. 145.

Atrophy of the tongue in a case of bulbar paralysis.

ciation difficulty is first noticed in articulating the letter i; then, in the course of time, r, sh, s, l, k, g, t, and usually last of all d and n. Of the muscles of the lip and lower face affected, the orbicularis oris is usually the first; then in less degree the levators of the upper lip, the quadratus triangularis, levator menti, and the buccinator. The eyelids are not usually affected—indeed, some excellent authorities say they never are; but I have had two cases in which there was

paresis of both upper lids. When the lips and lower facial muscles are implicated the expression of the face will undergo an alteration. The naso-labial folds deepen on each side, so that the patient has a woe-begone look, and the lower face is immobile, contrasting sharply with the animation given to the upper face by the intact muscles of the eyelids. Occasionally atrophy is demonstrable to a certain degree in the lips, but these muscles are normally so thin and small that it is usually difficult to determine this point. The patient finds difficulty in blowing, whistling, puckering the mouth, kissing, etc., and in pronouncing the lip-sounds, mainly noticeable with o, u, and a; then with p and f, and finally with b, m, and w. The combination ah seems never to be disturbed. Later on the palate becomes affected, and may be seen to be paretic. The pronunciation is then altered of the b and p, which are made to sound like me and we; this can be remedied momentarily by closing the nostrils with the fingers. The voice has a nasal twang. The laryngeal, respiratory, and cardiac symptoms are due to implication of the nucleus of the spinal accessory. It must be borne in mind, however, that the spinal accessory has two nuclei, one in the medulla oblongata, near the nucleus of the pneumogastric, and the other extending down into the cervical cord as far as the sixth or seventh cervical nerve. (See p. 85.) It is only the upper nucleus, in the medulla, that is affected in this disease. This has intimate connections with the pneumogastric, and from it various branches go to the nerve-trunk of the pneumogastric, to be distributed to the larynx, pharynx, and heart. The laryngeal, pharyngeal, and cardiac symptoms consist of an increasing paralysis of the muscles of these different organs that is evidenced: in the larynx, by affection of the vocal cords; in the pharynx, by the difficulties of deglutition spoken of; and in the heart, by alterations in the pulse and dyspnœa. It sometimes happens that the motor nucleus of the fifth pair is implicated, leading to paresis of the muscles of mastication. When this is the case the disease has an unfavorable prognosis. The patients are apt to suffer greatly from what seems like profuse salivation. In the majority of cases, however, this increase of the salivary secretion is purely apparent, and is due to the paresis of the buccal muscles preventing its retention within the buccal cavity in the normal manner; but occasionally there is a true hypersecretion.

PROGNOSIS. The prognosis in bulbar paralysis is a very serious one. It may be questioned whether a case has ever recovered. Long remissions of the symptoms have been observed by many writers, and I have seen the disease remain at a standstill for years, but ultimately death has come.

PATHOLOGY. The lesion is essentially one of degeneration of the ganglion-cell of the motor nuclei in the medulla oblongata. (See Plates II. and III.) The starting-point is usually in the nucleus of the hypoglossus; then the nucleus of the facial quickly becomes affected; and afterward the nuclei of the spinal accessory and the vagus. The spinal accessory nucleus here spoken of is the one in the medulla oblongata, and not the one which extends into the cervical cord, as has already been stated. The glosso-pharyngeal

nucleus is not always affected, that of the abducens very seldom, whilst the nuclei of the auditory and the trigeminus are always intact, except occasionally in the motor portion of the latter. The olivary bodies are almost always normal, and it is probable that the same is true of the formatio reticularis. The cerebellar peduncles are never involved. The pyramids are sometimes implicated, and there may be a descending degeneration of them. To the naked eye the medulla oblongata does not always display change. Not infrequently, however, there are a visible atrophy and a grayish color of the affected nerve-roots, generally most marked in the hypoglossal and the facial, less distinct in the spinal accessory, the vagus, and the glosso-pharyngeal. The changes in the muscles are similar to those found in progressive muscular atrophy.

In some cases no lesions whatever have been found in the medulla, and it may be surmised that the symptoms are then possibly due to implication of the centres in the cortex of the cerebrum, although it must be remembered that this is a mere supposition.

Diagnosis. The diagnosis is easily made. The disease can only be confounded with—

Facial paralysis;
The infantile type of progressive muscular atrophy;
Facial tropho-neurosis;
Affection of cortical centres.

From facial paralysis the diagnosis is easily made, because in this the onset is acute or subacute, the eye is wide open (lagophthalmus), the tongue is not atrophied or paralyzed, the affection is unilateral, and speech is affected but little and in a different manner.

In the infantile type of progressive muscular atrophy there is atrophy of other than the facial muscles, and the tongue and larynx are not involved.

In facial tropho-neurosis there is only a wasting of the face.

An affection of the cortical centres of the muscles of the lips, larynx, and tongue would have such symptoms as aphasia and paralysis of the arms and legs conjoined with it; besides, there would be the general symptoms of the various cerebral lesions.

Treatment. The treatment must be looked upon as palliative. It should consist of rest, some few drugs, electricity, and carefully prepared food.

Rest should consist of protection against atmospheric variations and total abstinence from mental or physical exertion. The cases in which I have seen long remissions have been housed in sunny, warm hospital wards, where they had the best of care and nourishment.

Some few drugs have been found temporarily useful, such as picrotoxin (gr. $\frac{1}{100}$ once to thrice daily), iodide of potash, and sulphate of quinine. Electricity should be applied in the form of both the galvanic and faradic currents, in the manner that has been described on page 123.

PROGRESSIVE OPHTHALMOPLEGIA.

Synonym: Poliencephalitis superior chronica (Wernicke).

HISTORY. The first description of this disease was given by v. Graefe in 1868. Gayet made his first autopsy in 1875, whilst Foerster in 1878 located the disease of exterior ophthalmoplegia in the floor of the aqueduct of Sylvius. Hutchinson and Gowers demonstrated in 1879 the lesion of the nerve-nuclei in a chronic case, and pointed out the analogy of progressive ophthalmoplegia with progressive bulbar paralysis and progressive muscular atrophy. Parinaud, Lichtheim, Erb, and Eisenlohr have also contributed to the subject, but the three most important communications of late years have been those of Wernicke, Mauthner, and Westphal. Wernicke has described the disease in his text-book under the head of chronic poliencephalitis superior, as the cells of the original nerves affected are in the gray matter of the upper part of the medulla oblongata in the fourth ventricle, the aqueduct of Sylvius, and the third ventricle (see Figs. 38 and 40), and he likens the disease to the myelitis affecting the cells in the anterior horns of the spinal cord. The posthumous monograph of Westphal was published in 1891, in which the autopsies made by the author are given.

DEFINITION. A paralysis, usually bilateral, of the levator palpebræ superioris and the muscles of the eye, either (1) of the so-called exterior or extrinsic muscles of the eye, or (2) of the so-called interior or intrinsic muscles of the eye, *i. e.*, the ciliary and accommodative muscles; or (3) both the exterior and the interior muscles simultaneously.

SYMPTOMS. The symptoms are those appertaining to a bilateral paralysis of all the different muscles of the eye, whether exterior or interior, as well as of the levator palpebræ superioris. It must be remembered that the third nerve—the motor oculi—supplies four of the exterior muscles of the eye: the internal rectus, turning the eye inward; the superior rectus and the inferior oblique, the two elevators of the eye; and the inferior rectus, one of the two depressors of the eye. This same nerve innervates also the ciliary muscles and the constrictor of the pupil, as well as the levator palpebræ superioris. Of the six exterior muscles of the eye, therefore, the third pair supplies four. Of the other two, one, the superior oblique, is innervated by the fourth or trochlear nerve; whilst another, the external rectus, has the sixth nerve, the abducens, going to it. Paralysis of the third pair, or motor oculi, is evidenced by ptosis, great retardation of the movement of the eyeball, and dilatation and immobility of the pupil. The ptosis is generally complete, although it is usually possible for the lids to be separated by the contraction of the frontalis muscle. If, therefore, this frontalis muscle be pressed upon strongly by the thumb, it will usually be impossible for the patient to separate the lids. There is upward strabismus of the eye, if the external rectus, deriving its nervous supply from the sixth nerve, be unimpaired, and the eye cannot be moved inward. Upward movement is impossible, while the movement downward takes place, if the superior oblique, supplied by the fourth nerve, be intact.

The dilatation and immobility of the pupil are due to paralysis of the sphincter pupillæ. This pupillary dilatation is not very great; indeed, many myopic eyes will present quite as much. It may therefore be necessary to determine the pathological nature of the dilatation by comparison with the pupil of the other eye, or by exclusion of any preceding ocular disease. But the characteristic of this moderately enlarged pupil is its complete immobility. It will not react to the light, either directly or indirectly, through the other eye. Certain fibres of the third pair go to the ciliary and accommodative muscles; *i. e.*, to the sphincter pupillæ and tensor of the choroid. These have, according to Hansen and Völkers, a different origin in the floor of the third ventricle from the other fibres of the third pair, and, as a matter of fact, paralysis of them alone is met with occasionally. In such a case not only will the moderately dilated and immobile pupil fail to react to light, either directly or indirectly, but it cannot be made to contract by any convergent movements of the eye or by any movement of accommodation. It has sometimes happened that the muscles of accommodation are intact, whilst the sphincter pupillæ has been completely paralyzed, or the pupillary reaction to light has been lost, whilst its reaction to accommodative movements has been preserved, or instead of a moderate dilatation there has been a very great dilatation, or the pupil has become very much contracted. The paralysis of the sixth nerve, the abducens, is manifested by an inward strabismus, due to paralysis of the external rectus. Paralysis of the fourth nerve, the trochlear, supplying the superior oblique muscle, is difficult to diagnose; the superior oblique draws the eye downward and outward, and inclines the upper end of the vertical meridian inward. But the reader had best read carefully the minute directions given for detecting paralysis of the different ocular muscles given on pp. 154, 155, and 156.

Headache may be absent in ophthalmoplegia, although it has been intense in some cases in which an implication of the sensory portion of the fifth pair was either found post mortem or suspected. It has been noticed that headache will disappear, as in cases of intracranial syphilis, upon the supervention of paralysis.

Nystagmus or nystagmus-like movements are occasionally seen. The disease is sometimes seen in conjunction with multiple sclerosis, locomotor ataxia, combined sclerosis, progressive paralysis, and such mental disturbances as belong to the psychoses, characterized by hallucinatory states of dread, hypochondria, delusions, and suicidal attempts. In these diseases the ophthalmoplegia may be a prodromal or concomitant affection. The progress of the disease is chronic, but it may be relatively so, first attacking one muscle or one group of muscles, then remaining quiescent, and again progressing with varying rapidity. It may be complicated with paralysis of the fifth pair, of the facial, with the general symptoms of bulbar paralysis, with locomotor ataxia, with progressive muscular atrophy, or with insanity.

Causation. The causes of ophthalmoplegia can scarcely be said to be known. It has been congenital, or it has developed a few days after birth, in infancy, and also in adults generally between forty and

seventy. Syphilis has been found to be a concomitant in a number of cases. The male sex is most often affected. Some of the cases have had a neurotic predisposition.

Pathology. The lesions of progressive ophthalmoplegia have been found to be disease of the cells in the upper part of the medulla oblongata which give origin to different nerves of the eye. The cells disappear, and the nerves to which they give origin degenerate through their peripheral filaments and into the muscle. In other cases there has been found to be a degeneration of the muscle- and the nerve-fibres, whilst the cells remain intact. In still other cases sclerotic foci affecting the nerves within the pons interrupt the nervous conductivity, although the muscle, the nerve, and the cell may remain intact. In one case of Kahler's there was an ependymitis with an underlying sclerosis. Finally, in some cases absolutely no lesion whatever has been found. But the most frequent form is that in which the cells are affected, the so-called nuclear paralysis, and the next most frequent form is that in which the nerves are the site of disease. When the cells are implicated, they are those of the abducens, the trochlear, and the oculomotor nerves. They may be degenerated, vacuolated, pigmented, or disappear entirely. Hyperæmia occurs occasionally, but it is not the rule. The ependyma, or lining of the ventricles, is occasionally proliferated. But of all these phenomena the degeneration of the ganglion-cells in various degrees, the decrease of the nerve-fibres running to the nucleus, and the implication of the surrounding parenchyma are the most usual factors, whilst hyperæmia and ependymitis are inconstant. From this it is evident that most cases are due to primary disease of the ganglion-cells, and not a hyperæmic or inflammatory process, as is maintained by Guyet, Wernicke, Thomsen, and Kowjewkneiff. As has already been said, however, a focus of sclerosis, in the course of the intra-medullary nerve-fibres may be the sole lesion, without the cells, nerves, or muscles being affected.

Prognosis. The prognosis of ophthalmoplegia is usually serious. In acute or subacute cases, commencing with great somnolence and general muscular weakness, the result is usually fatal. In acute non-complicated cases improvement may ensue, or the paralysis may become stationary, and sometimes the latter disappears. Indeed, symptoms of bulbar paralysis, if they be not attended by somnolence, do not exclude the possibility of cure.

Diagnosis. The diagnosis is easily made. The only question that can arise is as to whether the disease is complicated by other affections of the brain and the spinal cord.

Treatment. The treatment of ophthalmoplegia should consist of rest, tonics, stimulants, and electricity, as in myelitis.

The patient should be put to bed, if the attack be an acute one, until it is positive that the disease is not extending beyond the nuclei of the ocular nerves. In subacute cases, although it may not be necessary to put the patients to bed, they should be made to abstain from any mental or physical exertion.

For further details of treatment, the reader must consult "Myelitis."

CHAPTER V.

NEURO-MUSCULAR OR MUSCULAR DISEASES.

PSEUDO-MUSCULAR HYPERTROPHY.

Synonyms: Pseudo-hypertrophic muscular paralysis (Duchenne). Myosclerotic paralysis. Progressive muscular sclerosis. Atrophia musculorum lipomatosa. Lipomatous myoatrophy. Pseudo-hypertrophie der Muskeln.

DEFINITION. Pseudo-hypertrophic muscular paralysis is a chronic disease, usually causing paraplegia and great increase in size of the

FIG. 146.

Two children with pseudo-hypertrophic muscular paralysis, one (sitting) in the late stage, the other (standing) in the early.

lower limbs, occasionally affecting the upper extremities in the same manner, and without mental, sensory, rectal, vesical, or atrophic symptoms.

History. The disease was first really described by Duchenne, of Boulogne, in 1858, who denominated it "hypertrophic paraplegia of infancy," although Charles Bell in 1830, two Italian observers in 1838, Partridge in 1847, and Meryon in 1852, had put cases of it on record. Since Duchenne's description of it in his famous text-book, it has been generally recognized, but the best essays upon the subject have been by Gowers in 1879, Schultze in 1886, and Raymond in 1889.

Clinical History. The disease generally commences in early childhood with symptoms of weakness in the lower extremities, to which attention is first called by the patient's stumbling or falling frequently. As the malady progresses, the gait of the patient becomes characteristic, with legs spread wide apart, shoulders thrown back, and waddling; and it soon becomes evident that the lower

Fig. 147.

Case of pseudo-hypertrophic muscular paralysis, showing the difference in size of the upper and lower extremities, and the helplessness.

limbs are increasing in size. If the child is examined at this time, it will be found that it has a peculiar difficulty in getting up from the floor upon which it has been placed, or from a chair, or in going up stairs. He first puts his hands upon his knees, then grasps his thighs higher and higher, and so, as Gowers says, by climbing up his thighs he rises to the erect posture. Gowers considers this symptom pathognomonic, and I am inclined to agree with him. The muscles will also be found to be increased in size in varying degree, as is shown in Figs. 146, 147, and 148.

The upper extremities, and also the muscles of the neck and face,

are rarely affected. The hypertrophied muscles are weak, as can be determined by testing them in the proper way (p. 173). There are no sensory, mental, rectal, or vesical symptoms whatsoever. There is generally a lumbar lordosis, the spine being curved antero-posteriorly. The disease is a chronic one, lasting for many years, usually terminating, however, between twenty and twenty-five, and seldom continuing until forty years of age. Muscular atrophy is frequently conjoined with the muscular hypertrophy, and it is not unusual to find limited or general atrophy of the upper extremities in conjunction with hypertrophy of the lower. The patient generally goes on to a condition of perfect helplessness. The boy represented in Fig. 148, chubby and handsome and ruddy as he appears, is absolutely unable to move a muscle of his lower extremities from the trunk down, and has been for years in this helpless condition. Whilst the boy in Fig. 147 was not, when I saw him last, quite so helpless as this, he still was unable to get up from the floor or a chair without assistance. When death occurs, it is the result of intercurrent maladies which are greatly aggravated by the weakness of the trunk-muscles. Pulmonary affections are, therefore, especially dangerous to these individuals.

FIG. 148.

Case of pseudo-hypertrophic muscular paralysis, with absolute helplessness of hypertrophied lower extremities.

The difficulty in going up stairs is mainly due to the paresis of the knee and hip extensors, and the oscillating gait is probably also from the latter cause. As the patient throws his body laterally, so as to bring the centre of gravity over each foot, the weak gluteus medius cannot antagozize the inclination toward the leg that is off the ground. The difficulty in rising from the floor is thus explained by Gowers: The patient has not sufficient muscular strength to extend the knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power, applied between the fulcrum and the weight, acts at least advantage. He therefore places his hands on his knees, and his arms bring much of the weight of the upper part of the trunk on the femur close to the fulcrum, between this and the power, which acts, then, to greater advantage.

When the knees are extended the body can be raised into the upright position by the extensors of the hip, or by a push with the patient's hand. If these extensors are weak or paretic, the trunk is raised up by the patient grasping the thighs higher and higher. The antero-posterior curvature is due either to the weakness of the trunk-muscles or to that of the hip extensors, probably the latter, as a consequence of which there is an inclination forward of the pelvis, so that the upper part of the trunk has to be held back as a counter-poise. The lordosis disappears when the patient sits, and, indeed, it is then sometimes converted into a curvature in the opposite direction.

The hypertrophied muscles usually are normal in their electrical reactions until the disease is far advanced, when there may be decreased response to faradism and galvanism, but never any reaction of degeneration, so far at least as I have seen, although some French authors claim that this latter phenomenon occasionally occurs.

Causation. The disease is an hereditary one, and occurs principally in males and in early childhood. Some remarkable instances of the heredity have been published, as in Meryon's case, in which eight brothers died from the malady, whilst all the daughters escaped. Males are much more frequently affected than females. The disease first shows itself either when the child begins to walk, or when it is about four to seven years of age, in others about the tenth year, and rarely after this, although cases have been observed as late as the twentieth year. No other causes than these are known.

Prognosis. The prognosis of pseudo-hypertrophic muscular paralysis is very grave, as no case has ever been cured.

Pathology. The lesions of pseudo-hypertrophic muscular paralysis consist of a proliferation of the connective tissue between the muscular fibrillæ and the muscular bundles, subsequent fatty degeneration of this proliferated tissue, and atrophy or disappearance of the adjoining muscular fibres. The muscular atrophy is probably a secondary process, and results from the compression of the proliferated connective tissue, although there has been considerable doubt among pathologists as to whether there may not be a primary muscular atrophy in certain of these cases, whilst some authors actually classify pseudo-muscular hypertrophy among the muscular atrophies. Certainly in many cases, as I have already stated, muscular atrophy is observed in the same individual who presents the hypertrophy. The atrophy, whether primary or secondary, is generally a simple atrophy, the muscular fibres retaining their transverse marking to a late period, as well as their normal structure, although in certain cases there is a proliferation of the muscular nuclei, and granular, colloid, or waxy degeneration of the myolemma; in others there is actual hypertrophy of the muscular fibrillæ; whilst in still other instances alterations of muscle and connective tissue in some portions of the hypertrophied limb have been observed, and in other portions of the same limb simple atrophy, etc. In a number of autopsies which have been made some slight affections of the spinal cord have been shown, and it has been claimed that these

were in relation with the disease, but they either had been trifling or else were due to the action of the hardening-agents—so-called *artefacts*—or else to defective observation, as in Pekelharing's case, which was first described as presenting dilatation of the central canal, hyperæmia, and diminution in consistence of the gray substance, whilst the ganglion-cells of the anterior horns were supposed to have either disappeared or to be greatly degenerated, until the sections were examined by Schultze, of Heidelberg, who claims to have demonstrated that they came from a perfectly normal cord, in which there was no alteration whatsoever in the ganglion-cells. Middleton in especial has called attention to the danger of drawing conclusions from specimens hardened in alcohol, and narrates how he found in one such specimen, as he thought, a large quantity of round and irregular masses in the spinal cord, whilst a specimen from a section hardened in bichromate of ammonium was perfectly normal; nor were even the intramuscular nerve-fibres affected. (See Pathology, Chap. "Paretic Dementia.") For these reasons we are warranted in regarding pseudo-hypertrophic muscular paralysis as a peculiarly muscular affection, belonging to the class of the primitive myopathies.

Diagnosis. The diagnosis of pseudo-hypertrophic muscular paralysis is easily made, as a rule, by the history of the gradual onset in early childhood, by the difficulty of the child in rising from the floor or a chair, or ascending stairs; by the increased bulk of the muscles and their paresis; and by the absence of mental, sensory, rectal, and vesical symptoms, as well as by the lack of electrical alteration except in advanced cases. The diagnosis should be from

Progressive muscular atrophy; and
A muscular hypertrophy.

From progressive muscular atrophy pseudo-hypertrophic muscular paralysis can be distinguished by the fact that the former is seldom or never paraplegic in its onset; that the atrophy is first observed, and that even when hypertrophy is commingled with it, it is only so in individual muscles, and is of secondary importance.

Pseudo-hypertrophic muscular paralysis is differentiated from a muscular hypertrophy by the fact that in the latter rare disease the hypertrophy of muscle is attended by a corresponding increase in the muscular strength, just the opposite of what obtains in the disease that we are considering.

Treatment. The treatment of pseudo-hypertrophic muscular paralysis amounts to nothing, for the sad reason that no treatment whatever seems to be of any avail. Curiously, too, electricity, which acts so well in most spinal and muscular diseases, seems in many cases, if not always, to have an actually deleterious effect upon this malady. In the case represented in Fig. 147, for example, if the galvanic or faradic current was used in the earlier period of the disease, when the boy could still walk, its employment was always followed by perfect helplessness for a day or two; and I have ob-

served the same thing in several other cases. I have not been able of late years to treat a case of this disease in the way in which I should like to attempt it, because I have never been able positively to promise any relief; but from *à priori* reasons I have thought that it might be possible to treat the affected muscles in such a way as to increase the capillary circulation in them, at the same time that the absorbent action of the capillaries and lymphatics might be increased by sorbefacients. I should therefore use massage so gently and yet so efficiently that the blood would be made to pass in increased quantity through the muscles without any mechanical irritation of the muscular fibre from the hand of the person giving the massage. In my cases massage has always given great temporary relief, and patients have always been eager to have it continued. Then I should give the iodide of potash, in doses of 10 to 15 grains, three times a day, plentifully diluted in a full tumbler of water. Either with this or alone, I should use mercury in the form of a reliable oleate, and rub this over the affected limb, using a sufficient quantity to produce a slight ptyalism. I should have the massage given twice a day, and I should use the mercury once or twice a day. During the period of active treatment I should not permit the child to use the affected muscles at all, unless they showed some symptoms of returning power, and then I should permit voluntary use of the muscles very cautiously. Perhaps some one of my readers may have a chance to test my theoretic views in this terrible malady.

THOMSEN'S DISEASE.

Synonyms: Myotonia. Tonic spasms in voluntary muscles.

This disease was first described in 1876 by Dr. J. Thomsen, a physician of Schleswig, who was himself subject to it, and in whose family it had run for four generations. It had been referred to previously by Sir Charles Bell and Leyden. Since its description by Thomsen, a number of cases have been reported, and the best monograph on the subject is that by Erb, written in 1886.

It is a hereditary disease in the majority of cases, first appearing in early childhood, as soon as the child is able to make use of its muscles, although lately a so-called acquired form has been reported by Gowers, under the name of *ataxic paramyotone.* This case was in an adult of forty, but I have had under my care one in a child of six. Males seem to be more frequently affected than females. Of other causes we know very little.

The symptoms are a stiff and tonic spasm of the muscles, occurring upon voluntary exertion. This gives a peculiar gait to the children, which is quickly noticed. A little patient has innocently suggested for it the capital name of "the wiggles." Upon any exertion the muscles become very stiff. Usually all the muscles are affected excepting those of the face, tongue, and eyeballs, but in certain exceptional cases even these may be implicated. Eulenburg, however, has recently described what seems to be an identical disease, which he calls *congenital paramyotone,* and in which the facial muscles are

chiefly affected, especially the orbicularis oris and palpebrarum, so that the patient cannot speak or open the eyes in the paroxysm. Curiously enough, long-continued rest makes the disease worse, and the same effect will be produced by cold, heat, excitement, emotions, or sudden sensory impressions, whilst moderate exercise improves the muscular stiffness. The muscle is usually hypertrophied, although this is a false hypertrophy, as it is not conjoined with an increase of strength. The mechanical excitability of the muscles is increased, so that a blow upon them with a percussion-hammer will produce a slight contraction of the irritated fibres, which remain contracted for twenty to forty seconds. The faradic excitability of the muscles is also increased, a tonic persistent contraction resulting. With the galvanic current applied to the nerve or muscle, single shocks at the opening produce quick contractions. The muscles also show qualitative change with the galvanic currents, so that the contraction with the negative pole at the closure is equal in strength to the contraction with the positive pole at the closure, or the former may even be stronger than the latter. In some cases there are rhythmical, undulatory contractions moving from the negative pole to the positive pole.

The *pathology* of the disease is not known, as an autopsy has never been made, but the muscles have been excised or harpooned during life, and examination of them has shown that they are in a condition of enormous hypertrophy of all the fibres and great proliferation of nuclei, the transverse striation being indistinct; and there are also vacuoles in the muscular fibre and a slight increase of the perimysium. Dr. George W. Jacoby, of New York, has also called attention to the fact that the fibre is distinctly divided into angular fields, varying in size, and similar to the faintly indicated Cohnheim fields of normal muscle, and that there is almost everywhere, as seen with high power, a lack of continuity between the groups of sarcous elements.

The *prognosis* of Thomsen's disease is grave, inasmuch as we are not in a position to say whether a case of the congenital form can be cured. So far, in the thirty odd cases that have been reported, there has never been a cure, but it must be remembered that our knowledge of the affection dates back only to 1876. I have, however, seen what was perhaps a cure in an acquired case, with no return of the symptoms in three years. Patients, however, lead a fairly comfortable life, and death does not result from the disease itself.

The *treatment* consists simply of learning to avoid those things which have been found to have an unfavorable influence, as cold, damp air, excitement, etc. Electricity and drugs have not as yet proved to be of any value in the congenital type. My case, spoken of above, was cured by treatment with belladonna, rest, and massage. The drug was given at first one drop morning and evening, then three times daily.

CHAPTER VI.

INTRACRANIAL HEMORRHAGE, EMBOLISM, AND THROMBOSIS.

As the symptoms immediately resulting from lesion of the nerve-strands are almost identical in cases of hemorrhage, embolism, or thrombosis, and as the diagnosis between the three must be made by means of other than purely cerebral symptoms, I have considered it best to treat these three conditions in one chapter. Before entering more immediately into the subject, however, there should be a proper understanding of the way in which the cerebrum is supplied with blood. The researches of Heubner and Duret have thrown great light upon the arterial supply of the cerebrum, and the reader should master all the details of the cerebral circulation as described on pages 89 *et seq.*

Hemorrhage is the most common of all vascular disturbances of the cerebrum. The miliary aneurisms which were first described by Charcot and Bouchard are by far the most frequent cause of it. These little bodies vary in size from a pin's head to a millet-seed, are reddish in color, and are best demonstrated in a brain by washing away the cerebral substance with a stream of hot water from a tea-kettle, when, with care and patience, the whole arterial ramification can be laid bare. A section of the cerebrum may show simply little reddish dots, which an examination with a magnifying-glass will show to be larger than an unaltered arteriole. These miliary aneurisms are found upon the capillaries, whose diameter does not exceed that of one-tenth of a millimetre, the so-called capillaries of the first variety of Robin, but particularly in the capillaries of somewhat larger variety, which are visible to the naked eye, and which most anatomists call the arterioles or venules, although Robin designates them capillaries of the second or third variety. But they are also, though very rarely, found in the arteries of the base and the ramifications of these in the meninges. According to Andral and Durand Fardel, confirmed by Charcot, the parts which suffer most from hemorrhage are as follows, each part being named in the order of its frequency: the cerebrum (in which successively the most affected are the corpus striatum, the optic thalamus, the different lobes of the cortex, and the convolutions), the pons, and the cerebellum. There are certain portions of the brain in which hemorrhage seldom occurs, such as the tubercula quadrigemina, the crura cerebri, and the medulla oblongata, whilst it is scarcely ever observed in the cornu ammonis or the corpus callosum. Miliary aneurisms rarely occur before the age of forty, afterward with increasing frequency,

although cases have occasionally been reported in youth and childhood, especially one by Baillarger, and of late years there has been a growing tendency to recognize that infantile hemorrhage, probably due to this cause, is much more frequent than the older authors supposed. Next to miliary aneurism, a chronic endarteritis is the most common cause of hemorrhage; but it must be remembered that these two pathological conditions differ from each other. Miliary aneurisms often occur without the slightest atheromatous disease, just as marked atheromatous disease can take place without the formation of miliary aneurisms. Thus, in 77 cases collected by Charcot, miliary aneurisms were found in every instance, but 15 (over one-fifth) presented no atheromatous changes, whilst 25 of them (over one-third) were but slightly involved by atheromatous degeneration; and the remainder were atheromatous in varying degree. Miliary aneurisms are the result of a diffuse peri-arteritis of the small intracerebral vessels, sometimes associated with an atrophy of the walls of the large vessels of the base and the meninges. This peri-arteritis leads to thickening of the lymph-sheaths by proliferation of the nuclei of the latter and by the formation of fibrillary connective tissue. Alterations ensue in the muscular coat, consisting of a loss of the muscular fibres and disappearance of their transverse striping; but this muscular coat is always affected secondarily, generally where the alterations in the lymph-sheaths are most pronounced. In consequence of the loss of this muscular coat sacculated dilatation of the vessel occurs in places without alteration of the intima. Examined under a magnifying-glass, a miliary aneurism will be found to consist of the lymph-sheath and intima fused together, whilst the muscular coat is entirely wanting. Its contents are fluid blood, white blood-corpuscles, fat-granules, granular bodies, and amorphous masses. When a hemorrhage has occurred it is at first a dark, almost black clot, but sometimes in disease altering the constituents of the blood, as in pyæmia, it is not coagulated. The wall of the hemorrhagic focus is colored a bright red, which is due to capillary hemorrhages. The gray matter of the basal ganglia offers less resistance than the centrum ovale to the hemorrhage. The clot becomes gradually altered to a dark brown or chocolate color, thence becoming yellowish-red and yellow. At first the surrounding tissue is œdematous and pultaceous. This hemorrhagic focus may become encapsulated by a thin layer of thickened neuroglia, occasionally even by the formation of actual fibrillary connective tissue. A so-called apoplectic cyst may be formed—*i. e.*, the fibrin of the clot and the injured nervous elements undergo a retrograde fatty metamorphosis and are gradually absorbed by the lymphatics, so that a cavity remains, filled with clear or colored fluid, and surrounded by a smooth wall; or the surrounding œdema may extend and cause death. It is often extremely difficult, however, in this last stage to distinguish an apoplectic cyst from one that has resulted from softening. At other times the walls of the encapsulated clot become approximated before the central portions of the clot have been absorbed, and there is thus formed the so-called apoplectic cicatrix.

Increase of arterial tension may be a cause of cerebral hemorrhage, although it has never yet been shown that this can occur in arteries that are not diseased. In the general endarteritis which is associated with hypertrophy of the left ventricle and a contracted kidney, associated with endarteritis of the intracranial vessels, hemorrhage is by no means infrequent. Fatty degeneration of arteries is also a frequent cause, either alone or by the endarteritis that results in atheroma, although this latter is more usually an indirect cause by rendering the walls of the larger vessels rigid, so that the blood streams into the arterioles without the regulating influence of the normal contractile vascular coats.

The red softening which was so frequently described by the older writers, and which was by them supposed to be a primary process of softening, is now regarded rather as secondary to the arterial disease. Intracranial hemorrhage also occurs in various diseases which lead to changes in the tissue of the blood, such as pyæmia, typhoid, scorbutus, puerperal fever, chlorosis, leucocythæmia, pernicious anæmia, and icterus. Heredity also plays a certain part in causing hemorrhage by inducing the tendency to arterial disease, so that it is well established that certain families have a predisposition to this condition. The short neck, broad chest, and florid complexion, however, which were formerly supposed to indicate a tendency to hemorrhage, are no longer regarded as of any prodromal value; indeed, it is certain that no particular bodily type is known to predispose. Men are somewhat more affected than women. Altitude also appears to predispose, as it is more frequent in elevated regions, and I have known instances in which it has occurred while passing over lofty mountain chains. Alcohol also predisposes by producing fatty degeneration of the vessels and premature endarteritis.

Cerebral emboli may consist of fibrinous masses from the left cavities of the heart, the aortic and mitral valves, the arch of the aorta, and the pulmonary veins, or from a thrombosis in the cerebral vessels. After certain acute and exhausting diseases the blood may become so altered in quality as to be prone to coagulate, or, as in atheromatous and calcareous disease, the roughened coats of the vessels may whip up the fibrin. In certain cases of malaria and intermittent fever the cerebral capillaries may be plugged up by pigment-emboli. In other cases they may be obstructed by drops of fat from atheromatous formations in the larger vessels, or, after injuries to bone, the fatty tissue of the marrow may be carried into the capillaries. Virchow has described what he calls a lime-metastasis, which is due to lime being deposited on the walls of the capillaries when there is some associated bony disease.

Clinical Symptoms. In all cases of cerebral hemorrhage, embolism, or thrombosis distinction must be made between the reflex symptoms and the localizing ones.

The reflex symptoms are due to the shock which the cerebrum as a whole sustains from the outpouring of blood of a hemorrhage, or from the plugging of a vessel by an embolism or thrombosis. They consist of loss of consciousness or coma, mental disturbances, con-

vulsions, and alterations of temperature. When there is a loss of consciousness, the onset is a so-called apoplectiform one, and this loss of consciousness may be sudden and complete, or gradual, and in the latter case it is usually preceded by some abnormal sensations about the head, by confusion, difficulty of speech, drowsiness, nausea, or vomiting. When the patient is comatose, the face is usually flushed and swollen, or pale and clammy, the eyes fixed, the pupils widely dilated and sluggish, the respiration slow, sometimes stertorous, with cheeks blown out in respiration, pulse generally slow and full, and the limbs are inert. In the attack with convulsions the latter are generalized, or confined to the side that is afterward found to be paralyzed.

The temperature is subnormal in almost all cases, going down sometimes to $96\frac{5}{10}°$ F., but it rises markedly in the non-fatal cases within the first twenty-four hours, and in fatal cases may run up to as high as 108°, although usually it oscillates irregularly between 99° and 100° or 101°.

Conjugate deviation of the eyes, with rotation of the head away from the paralyzed side and toward the hemisphere which is affected, often occurs as a temporary symptom. Alternate conjugate deviation is that condition in which the eyes turn first to one side—generally toward the lesion—and then to the other. It is supposed to indicate a cortical or pons lesion; and it is believed that the deviation is toward the lesion when the brain is affected, and the other way when the pons is implicated. The eyes are usually fixed, but there is occasionally slight nystagmus.

The localizing symptoms will vary according to the location of the disease, but the most frequent site is in the internal capsule, at the anterior portion just around the genu, where the motor fibres come down from the hemispheres (*vide* pp. 59 and 145, and Fig. 35), and this gives rise to symptoms of hemiplegia upon the opposite side of the body, although in cases of non-decussation of the motor fibres the symptoms may be upon the same side, as Brown-Séquard has pointed out. In these cases the patient is afflicted with paralysis of the lower part of the face (see p. 85) and of the upper and lower extremities, whilst the speech is usually implicated also. An examination of such a patient will show that the naso-labial fold upon the affected side is flattened out, so that the face is drawn to the other side, the tongue deviates toward the affected side, and the arm and leg on the same side with the face are impaired in strength, and in certain cases also in the sensations of touch and pain, more rarely in muscular sense, least frequently in temperature-sense. The tendon and cutaneous reflexes upon this side, notably the knee-jerk, are temporarily diminished or abolished, and this may often be a diagnostic sign of great value, especially in the cerebral hemorrhage that takes place with uræmic coma. Immediately after an attack the paralyzed limbs are often warmer and redder than upon the other side, and they may become swollen from subcutaneous œdema, or it may even happen that some of the joints may be subacutely inflamed. Occasionally, though rarely, the paralyzed limbs may

atrophy. In the unfavorable cases contracture of the affected limbs may gradually result, and this contracture may be early or tardy, coming on immediately after the attack or some time after. In either case this contracture is a sign of ill omen, but more especially so when it follows at an early date. Even when it comes late, however, it is seldom recovered from. An acute bedsore may form in certain fatal cases over the centre of the gluteal region on the paralyzed side, and is of very evil significance, although not in my experience necessarily fatal. In cases of hemorrhage in children the growth of the whole limb may be arrested. (See Figs. 149 and 150.) In the majority of cases of cerebral hemorrhage occurring in this region of the internal capsule, the symptoms are generally those that have been described, and were detailed by the older authors under the name of apoplexy (a name still retained by the laity), and which most of the text-books of to-day present under the title of hemiplegia—although, really, hemiplegia can result from hemorrhages into so many different parts of the brain that it is unscientific to apply the term to lesion of any one particular locality.

When hemorrhage occurs into the convolutions constituting the motor area (*vide* p. 139) there is more apt to be paralysis of one limb (so-called monoplegia), or of the two limbs, or of the upper extremity, with coincident speech-defect. As will be seen by Fig. 35, a hemorrhage of the motor area sufficient to cause hemiplegia would have to be spread over so large a surface as to make its occurrence extremely infrequent, except from trauma.

Hemorrhage into the occipital lobe, the lingual or fusiform lobule, or the angular gyrus, would give rise to symptoms of hemianopsia (*vide* p. 150).

Hemorrhage into the posterior portion of the first and second temporal convolutions would cause word-deafness (*vide* p. 140).

Hemorrhage into the cerebellum would give rise to symptoms that are detailed upon p. 149.

The symptoms, indeed, of hemorrhages into the different regions of the cerebro-spinal axis have been so fully considered under "Localizations," pp. 139 *et seq.*, that it is needless to consider them again.

Prognosis. The prognosis of intracranial hemorrhage will vary according to the locality affected and the severity of the symptoms. When the coma is profound, quickly followed by a rising temperature and acute bedsores, the case will probably have a fatal termination. When contracture supervenes, either early or late, the prognosis as to recovery from the paralysis is bad, as is also the case when marked atrophy supervenes in a few weeks.

Hemorrhage into the internal capsule is always a serious symptom, even when there is no rise of temperature or contracture following, and this is especially so when it occurs in the course of Bright's disease. Hemorrhage into the cortex is not necessarily of such bad omen, because of the free anastomosis of the cortical capillaries which has been described by Heubner and Duret; but even then, when the hemorrhage is into the motor area and is followed by marked

contractures and atrophies, it is not recovered from. Of the prognosis of hemorrhage into the cuneus, the occipital convolutions, the lingual and fusiform lobules, and the angular gyrus (causing hemianopsia), we know very little. The few cases that have been reported of hemorrhage into the lemniscus have been fatal, and this is usually the case with hemorrhage into the pons. Cerebellar hemorrhage has not been accurately studied, because of the difficulties of cerebellar localization; but of the cases which have been studied, most have resulted fatally.

Diagnosis. The diagnosis of cerebral embolism can usually be made by ascertaining the causes which can produce cerebral embolism, such as cardiac lesions and pulmonary lesions; but it may happen that an embolus may result from a washed-off thrombus of some larger intracranial vessel being carried to a capillary, and in such a case the diagnosis cannot be made. It is usually stated in the text-books that a diagnosis between embolism, thrombosis, and hemorrhage can be made by means of the clinical symptoms. There is not only inaccuracy of observation about this statement, but also some confusion of thought. What the writers have meant to say is that the diagnosis can be made in these three conditions by observing the onset, which in cases of embolism will be sudden, and in cases of thrombosis and hemorrhage will be more gradual. But even this statement is inaccurate. An embolus, for instance, may enter a capillary that it does not completely plug up at once, so that the onset of the symptoms may be gradual, or it may fit the capillary so accurately as to plug it up suddenly, in which case the symptoms will be sudden. A thrombosis, on the other hand, may form so very gradually as to produce no symptoms until, by the superimposition upon it of layers of fibrin, the final moment of occlusion of the vessel has been reached, when it may plug up the vessel suddenly and completely, and the symptoms would be sudden also. A hemorrhage may be produced by a sudden or gradual rupture of a vessel, so that the symptoms may be either sudden or gradual. I have made a number of post-mortems in which I satisfied myself of the impossibility of making a diagnosis between embolism, thrombosis, and hemorrhage by means of the suddenness or gradualness of the symptoms. I am therefore positive that it is utterly impossible to make a diagnosis between the three conditions in this way. Usually, therefore, the first thing to be sought for will be the cardiac or pulmonary lesions that might give rise to emboli. If they are found, and if the onset has been sudden, a diagnosis of embolism is probable, although these cardiac and pulmonary conditions may exist without causing emboli. If these cardiac and pulmonary sources of embolism are absent, however, it is utterly impossible to make a diagnosis between embolism, thrombosis, and hemorrhage, and all the diagnosis that we can make is between arterial lesions and other lesions of the brain that might produce similar clinical symptoms.

The latter conditions are—

Tumor;

Fractures;

Thrombosis of the cerebral sinuses;
Ménière's disease;
Cerebral palsies of childhood;
Intracranial syphilis.

Intracranial tumors are very easily differentiated from cerebral hemorrhage, thrombosis, or embolism, because they do not have an acute onset, are preceded by headache, various localized paralyses, changes in the optic disc, and occasionally by mental alterations.

Fractures of the skull should never be confounded with cerebral hemorrhage, except in cases that are picked up in the streets without an antecedent history. The hemorrhage of these fractures is much more apt to be meningeal than into the substance of the cerebrum or cerebellum. Usually the history of trauma, the detection of fractured bone, the outpouring of bloody serum from the ear in fractures of the base, etc., are symptoms which will make the diagnosis clear.

Thrombosis of the cerebral sinuses may occur from marasmus in children in the first six months of life after severe diarrhœa, or in adults from pyæmia, cancer, senile marasmus, or other debilitating causes, or from diseases of the cranial bones which may follow traumata, erysipelas, or furuncle; but it is impossible to make the diagnosis by means of the symptoms alone, although this may sometimes be provisionally done by the conjunction of the symptoms and the presence of one of these known causes. Otitis media or interna may also be a cause. The symptoms are varied, and consist of coma, convulsions, strabismus, contractures, tremors, and paralyses, these paralyses and tremors being either one-sided or bilateral; and these symptoms should always be regarded with suspicion when they supervene upon diseases of the internal ear. (See Chap. "Venous Thrombosis.")

Ménière's disease of the true type should never be confounded with hemorrhage, although it was frequently in former years. The loud noises in the ear, like pistolshots or bells, the partial loss of consciousness, the turning in certain directions, and the absolute absence of any paralytic symptoms whatsoever, need only to be studied and observed in order to make the diagnosis (*vide* Chap. XV., "Ménière's Disease").

The cerebral palsies of childhood are due to many other causes than cerebral hemorrhage (see Chap. "Cerebral Palsies of Childhood"), and it is only when there is a hemiplegia coming on after such symptoms as we see in the adult that we are warranted in making a diagnosis of cerebral hemorrhage. The paraplegia, the peculiar contractures, the positions of the limbs, the marked mental symptoms, are usually entirely different in their distribution from what is seen in the adult from cerebral hemorrhage.

Intracranial syphilis can in many cases, as I have demonstrated, be diagnosed by means of the quasi-periodical headache with obstinate insomnia, which ceases upon the supervention of paralytic or convulsive symptoms; and when the headache and insomnia are followed by hemiplegia in an individual under forty years of age the diagnosis may be made with positiveness of intracranial syphilis.

In any case of hemiplegia, however, occurring under forty years of age, syphilis should be suspected, even though there has not been a history of headache and insomnia. In some cases of intracranial syphilis, too, the hemiplegia has been preceded by mental dulness for weeks beforehand to an extent which is never seen in ordinary cerebral hemorrhage such as we are describing. The forms of intracranial syphilis which are due to a gummatous meningitis at the base of the brain are attended by lesions of the cranial nerves, as well as by paralytic symptoms of the extremities.

TREATMENT. In all cases of cerebral hemorrhage the patient should be kept quietly in bed until all symptoms of reflex disturbance of the cerebrum or cerebellum have disappeared. The diet during this period should be simple and moderate in quantity, consisting mainly of eggs, soups, fish, and milk. The patient should not be allowed to rise from a prone position. If there is any tendency to convulsions, moderate doses of the bromide of potash should be administered, 10 grains once, twice, or even three times a day. I have seen excellent results from venesection, and I believe that this old remedy has been allowed to pass into too great disuse, mainly in deference to the prejudice of the laity. I should have no hesitation whatever in taking ten or twenty ounces of blood from a patient evidently full-blooded, and who could therefore bear its loss well, because the immediate results are, as I have said, sometimes startlingly beneficial. In such a case, if venesection cannot be performed, brisk purgation with saline cathartics, or with croton oil, one drop given once or twice, will often be found of advantage, although neither of these drugs will equal the bloodletting in suitable cases. As the patient begins to recover from the reflex disturbances small doses of the bichloride of mercury, $\frac{1}{32}$ grain three times a day, have seemed to me to be often of considerable value, although, of course, it is a difficult matter to speak positively about the effect of this medicament, as the improvement may have been due to natural causes. In the same spirit I would commend the use of moderate doses of iodide of potash, 10 grains three times a day, although I do not think that it is as efficacious as the mercury. Of late years trephining has been recommended in cases of intracranial hemorrhage. I cannot say that I view this procedure with any great favor, and this for several reasons. In the first place, as I have already stated, it is often extremely difficult to make the diagnosis between hemorrhage, embolism, and thrombosis. In the second place, the hemorrhage is most likely to occur into the internal capsule, and it is very difficult to reach this portion of the brain with accuracy so as to hit exactly the site of the hemorrhage; and even if this be done, it is very questionable whether the puncture with the needle would not inflict more damage upon the delicate interwoven strands of this locality than the hemorrhage itself would do. Should the hemorrhage, however, be subcortical in the centrum ovale between the cortex and the basal ganglia, it would be almost impossible to localize it, and the wild groping with the needle, such as we have so much difficulty with in cases of cerebral tumor, would be certain to inflict more damage than

the hemorrhage itself. If, however, the hemorrhage can be positively diagnosed in the cortex, I can see no objection to this method of relief. When the individual is able to leave his prone position the paralysis of muscle, if the lesion in the brain has been such as to cause this, may undoubtedly be favorably affected by the use of the faradic current, brushing the electrodes up and down the limbs, passing a gentle current through the different muscular groups, and doing this two or three times a week for a period of several months. This simple application of electricity can be done quite as well at the house by some member of the family with a small faradic battery. General tonic medication will oftentimes be of use, too—a fact which is often overlooked. The best of these is some of the quinine preparations, preferably the elixir of calisaya bark.

CHAPTER VII.

DISEASES OF THE CEREBRUM.

CEREBRAL PALSIES OF CHILDHOOD.

Definition. These palsies consist of hemiplegia, double hemiplegia (diplegia), or paraplegia, with mental defect, spastic symptoms, and atrophy that is generally slight and without marked alterations in electrical reaction.

History. The first authors who portrayed what is nowadays known as porencephaly were Braschet, in 1831; Lallemand, in 1834; Rokitansky, in 1835; and Cruveilhier, in 1849; although some of the cerebral lesions of the infant had been described by Reil, in 1812; Cazauvielh, in 1827; and Billard, in 1828. But the first accurate description of paralysis of cerebral origin in children was that of Henoch, in 1842, which was supplemented by Little, in 1853; Turner, in 1856; and Von Heine, in 1860. Heschl formulated the term "porencephaly," in 1859, to designate the losses of cerebral substance. From that time to the present day the literature of the subject has been very abundant, and the most prominent among the contributors have been Leidesdorf and Stricker, in 1865, on the production of encephalitis in chickens, Tigges concerning encephalitis in rabbits, Virchow, in 1865, on congenital encephalitis of a peculiar nature; Cotard, in his *thèse de Paris*, in 1868; Kundrat, in his great book on Porencephaly, in 1882; Parrot, in 1883, on Steatosis; Strümpell, in 1844, upon what he calls Polioencephalitis, and, amongst the latest authors, Gandard, Jendrássik and Marie, McNutt, Wallenberg, Knapp, Audry, Gibney, Gowers, Osler, Sachs, and Peterson.

Etiology. The causation is by no means certain. Many attendant or preceding diseases, many circumstances of immediate environment, and many hereditary peculiarities, are ranked in the time-honored way among the causes, without scientific warrant, however, although each and all may be factors in certain cases, bearing the same relation to this class of lesions that they do to so many other diseases. In this way great stress has been laid upon marriages of consanguinity; syphilis and intemperance in the parents; difficult delivery; asphyxia of the newborn; cerebral traumata; infectious diseases, such as pertussis, typhus, and variola; abnormal conditions of the mother during pregnancy; violent vomiting and defective nutrition. All these cases are either congenital, having occurred during fœtal life, or have their onset in the first three years of life. The paraplegiæ are generally congenital, whilst the double hemiplegiæ (diplegiæ) usually occur during the first three years of life.

Clinical Symptoms. In all diseases of the intracranial contents, whether occurring in the child or in the adult, as has been elsewhere said, distinction must be made between the symptoms which are reflex and those that are direct or localizing. For example, a lesion of sudden onset, such as a hemorrhage, a thrombosis, or an embolism, may impinge upon the leg-centre of the cortex, primarily causing unconsciousness or a convulsion, fever, etc., which symptoms pass off, and there is detected a paralysis in the leg that has been overlooked or obscure. The reflex symptoms are due to shock of the whole cerebral substance from a sudden molecular change, whilst the direct or localizing ones are caused by actual destruction of a certain area of the brain-tissue. These considerations, too, apply especially to the cerebral affections of children, who are more liable to reflex disturbance than adults.

The reflex symptoms are convulsions, fever, delirium, hebetude or coma, and emesis.

The convulsions are not pathognomonic. They may be general, or they may implicate one member, the limbs on one side, or certain muscles alone. They may be tonic or clonic. It has been a growing doctrine of late years that cortical lesions in the adult are attended by convulsions, while subcortical ones are not so accompanied; but however true this rule may be in adults, it is certainly not so of children, in whom unconsciousness is not infrequently observed in poliomyelitis or even from reflex causes. The fever seldom goes above 101° or 102° F., and often there is no fever at all, although it must be remembered that in this particular we must usually rely upon the statements of parents or relatives. There is generally no delirium, and when there is, it is slight and temporary. Hebetude is present, as a rule, and coma is sometimes observed, although both of these conditions, like fever, may be entirely absent.

Emesis seldom occurs, although the child may not take food more because of the hebetude or coma or the general malaise than from nausea.

The localizing symptoms will depend upon the area of the brain implicated.

So far as they have been determined, however, the localizing symptoms are paralyses, muscular wasting, exaggerated tendon-reflexes, contractures, mutism, mental impairment, and speech-defects; although most of the cases have a preponderance of motor symptoms.

A classification of these paralyses by the motor symptoms is as follows:

Hemiplegia;
Bilateral hemiplegia or diplegia;
Paraplegia.

Hemiplegia generally occurs during the first three years of life. In such cases the face is not always affected, and when it is, the upper portion (the eyelids and brow) is not involved. Figs. 149 and 150 represent a hemiplegia which had its onset in a boy five years before the photograph was taken. His face is keen and intelligent, and the only mental defect in his case was that he was good-naturedly

uncontrollable and difficult to teach for about two years after the onset; but since this period he has been as intelligent as the other children of the same family—an excellent test, by-the-by, of a child's mental stature.

FIG. 149.

(FRONT VIEW.)

FIG. 150.

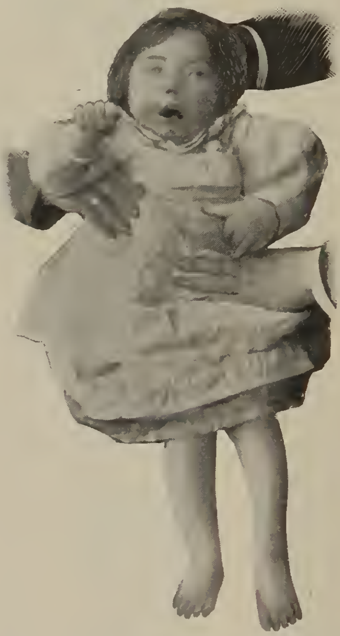
(REAR VIEW.)

Hemiplegic boy of eight, showing difference between right (affected) and left (unaffected) sides five years after onset.

The name of birth-palsies has been suggested by Gowers for congenital bilateral hemiplegia or diplegia which results from prenatal causes or accidents occurring through excessive pressure of the brain in parturition. Fig. 151 represents such a case, with marked mental defect, which is very evident in the child's vacant expression.

Paraplegia is usually a disease of early infantile life or is referable to abnormal conditions of the mother during pregnancy. Contracture of the paralyzed muscles is usually present in all these three forms, few exceptions to this rule being observed. When limbs thus affected are subjected to movement, they oppose a soft, wax-like resistance, slowly yielding, and returning gradually to their former position when the compelling-force is removed. Often there is a bar-like rigidity of the lower extremities, which is well represented in Fig. 151. This can be easily evoked by handling the limb,

Fig. 151.



Congenital diplegia, with marked mental defect.

dangling it to and fro for a few seconds, or tapping steadily on the quadriceps tendon. The extensor muscles are generally less affected than the flexor. In many cases the small joints are particularly pliable, so that the fingers can be moved in any direction, either extended, flexed, or abducted and adducted with extreme facility. A lock-like movement will be observed when a limb is suddenly and quickly flexed. (*Vide* page 169.) Various muscular deformities result from these contractures. As a general thing, the tendon-reflexes are exaggerated in the limbs affected, but not to any great extent. Varying degrees of mental impairment take place, ranging from a slight feebleness of intellect to idiocy of the most pronounced type. (Figs. 151 and 152.)

The wasting of muscles is not so marked as in poliomyelitis anterior, being more in the form of arrested development. One little patient

under my care shows each year a greater difference in the size of the arms, the affected one being relatively smaller, owing to the fact that no development takes place in it, while the other is constantly growing. Speech-defects and mutism are of common occurrence. No marked degree of disturbance of sensation is usually noted, but it must be borne in mind that in infants and young children it is impossible accurately to test the sensations. Defective vasomotor circulation and lower temperature of the affected limb are of frequent occurrence. The electrical reactions are normal, except that there is a slight quantitative increase to faradism, and also to galvanism in some instances. Weir Mitchell has described in the hemiplegic form, according to Osler, one case in which post-hemiplegic tremor affected the entire arm, twenty-four cases of hemiplegic chorea (or hemi-

FIG. 152.

Face with cerebral palsy of congenital origin.

ataxia), athetosis and mobile spasm in six cases. So-called spastic chorea and double athetosis have been described in the bilateral hemiplegias. Epilepsy often occurs in these unfortunate cases, as, out of one hundred and forty cases reported by Sachs, the percentage was $44\frac{3}{10}$. There were forty-one cases of general epilepsy among the hemiplegias, one case of petit mal, and nine of the Jacksonian type, about 50 per cent. in all. In twenty-four cases of diplegia, one had Jacksonian epilepsy, and seven (29 per cent.) had general epilepsy. Four cases of general epilepsy (about 36 per cent.) occurred in eleven cases of paraplegia. The percentage in these cases differs very little from that given by Wallenberg, Gandard, and Osler.

DIAGNOSIS. The diagnosis should be made from the following diseases:

Traumatic meningitis;
Cerebro-spinal meningitis, of epidemic or sporadic origin;
Suppurative meningitis;
Tubercular meningitis;
Transverse myelitis;
Myelitis of the anterior cornua;
Intracranial tumors;

Hydrocephalus;
Reflex hebetude, coma, or delirium.

A careful history should always be sought for in all such cases from some intelligent person—the mother generally the best, as having been constantly with the child from the onset, unless the disease has been recognized by some intelligent physician early in its course. The diagnosis is usually made very difficult by the lack of this precise history.

A diagnosis of traumatic meningitis can only be made when the trauma is positively known.

In cerebro-spinal meningitis of epidemic or sporadic origin the disease is recognized by the characteristic retraction of the head, by the fact that the disease is known to be prevalent at the time, by the ages of those attacked being mostly between one and fifteen, by the fact that the paralysis is usually one-sided and rarely paraplegia or double hemiplegia, by less tendency to permanent mental impairment, and by marked tendency to hebetude and coma.

Suppurations in the nasal cavities, orbit, ear, or lung, or some septic condition, mark the presence of suppurative meningitis. Also, a fluctuating temperature, rigors, or chills are apt to prevail, and there is a peculiar remission in the symptoms.

The chronicity of the cerebral symptoms and the infrequency of an acute onset will aid in the diagnosis of tubercular meningitis, coupled with a history of tuberculosis in either the patient or the family.

Children are rarely subject to transverse myelitis, but when it does occur it causes paraplegia, perhaps bedsore, as well as rectal and vesical paralysis. There is no mental impairment, and usually no affection of the upper extremities.

Myelitis of the anterior horns, as is indicated by the name, is an affection of the anterior cornua of the spinal cord, causing a sudden loss of the ganglion-cells, characterized by absence of knee-jerk, flaccid paralysis, atrophy of certain muscular groups, usually in one limb, and altered electrical reactions. It is very seldom hemiplegic in the child, and never doubly hemiplegic, but necessarily monoplegic in its distribution. There may be a doubt as to the diagnosis for a few days in cases that are marked by convulsions, hebetude, coma, and fever, but the question may be settled by the ultimate distribution of the paralysis.

Intracranial tumor is extremely difficult to differentiate from cerebral palsy. But in the former the tendency is to a greater localization of the symptoms, to chronicity, to a greater frequency of purposeless reflex so-called cerebral vomiting, and to neuro-retinitis.

Hydrocephalus is almost positively diagnosed by the peculiarly shaped head, and is often associated with tubercular meningitis.

Hebetude or coma, seemingly of a most alarming nature, may prevail for a few days in children, but will soon disappear, being apparently caused by a reflex indigestion or vasomotor condition. In the acute febrile affections grave conditions of hebetude, coma, or delirium may often occur, and it is a matter of great nicety to deter-

mine what proportion is due to implication of the cerebrum and its membranes, and how much is reflex from the primary disease.

PATHOLOGY.—Of the numerous diseases of the infantile brain, those are well known that are due to a suppurative meningitis occurring from a distinct cause—cerebro-spinal meningitis of the sporadic or epidemic type and cerebral meningitis of traumatic origin. Outside of these, however, the many pathological conditions affecting the fœtal and infantile brain are involved in great obscurity. It is not strange that this should be so. Almost all these lesions are chronic; autopsies are not had until a considerable time after the onset, so that the terminal conditions—the post-funereal ones, so to speak—are the only ones that can be studied, and a bewildering diversity of opinion necessarily obtains as to the exact nature and origin of the causative processes. Nor has the clinical expression of these intracranial processes thrown much light upon the subject. It must be remembered that the brain is a highly complex structure. In the child only gross impairment of motion, sight, and hearing can be observed, and even these only after a certain age. The ordinary forms of sensation, the group of symptoms included under the common name of aphasia, the so-called word-deafness, the fine muscular movements, the more delicate impairments of sight, such as hemianopsia, etc., the subjective sensations of vertigo, and the peculiar noises that are often of so much diagnostic importance in certain impairments of hearing—all these are finer functions that cannot be tested in the child until it has attained nearly the intelligence of youth, and they are entirely beyond our recognition in the infant. Moreover, these old lesions that have begun in infancy or fœtal life do not cause the same localized impairment of function that they do in the adult brain. Hydrocephalus, hemorrhage, embolism, and thrombosis are described in their respective chapters. Aside from these, the following lesions have been observed in the brains of fœtuses and infants who had presented during life the clinical symptoms of the cerebral palsies:

Encephalitis and meningo-encephalitis;
The so-called polio-encephalitis;
The congenital encephalitis of Virchow;
Porencephaly:
Apoplexy of the newborn;
Thrombosis of the cerebral veins;
Atrophy and sclerosis.

Encephalitis and meningo-encephalitis generally begin in the pia and cortex, and proceed with cellular infiltration and sclerosis, minute hemorrhages, eventually atrophy, or, more rarely, hypertrophy of the affected convolutions. The large basal ganglia and even the spinal cord may be implicated simultaneously in this process, but this is to be distinguished from the secondary descending degeneration which may result from a lesion in the cortex. The onset of these processes has not been studied microscopically. The thick connective-tissue masses may vary in size from that of the cortical surface of the whole hemisphere to a mere cicatricial streak.

In these sclerotic masses scanty remains of nerve-elements are found. The connective tissue may consist of a network, in the meshes of which are seen scanty openings for the vessels and granulated cells and nucleoli, or it may be very dense, and the openings for the bloodvessels large and numerous. Because of the seeming relationship between the density of the connective tissue and the size of the vascular openings, because Hayem has found the walls of the bloodvessels thickened, especially the adventitia, and because Marie has found these vascular walls in a condition of inflammatory infiltration, with embryonic nuclei, it has been suggested that the vascular lesions were the initiative process; but there is nothing to show whether or not they are the cause, effect, or part of a general underlying condition. In the hypertrophic form of sclerosis the cerebral substance is not infrequently dotted with ten or twelve masses, varying in size from that of a bean to that of a ten-cent piece, round or streak-like, or of the consistence of India-rubber, principally in the cortex or in the central ganglia. From these masses the pia tears easily, but is adherent to the surrounding tissues, which are of a reddish color from a markedly vascular injection. These masses consist of thick connective tissue, with spindle-cells, and pass into the normal tissue without sharp demarcation. A few atrophied ganglion-cells are found, filled with pigment and granules.

In 1884 Strümpell advanced the theory that there was in children an acute infectious encephalitis of the motor convolutions, analogous to the acute myelitis of the anterior cornua of the spinal cord. This seems to me to have been one of the most flippant pathological suggestions ever made in medicine, advanced without the slightest proof on the part of its author; and as yet only one writer, Ranke, has adopted this view, and only one clinical observation, probably merely coincidental, not elucidated by an autopsy, has been cited in support of it. The avidity with which the suggestion has been seized for discussion and observation, and the large literature which has grown up in refutation of it, are pitiable illustrations of the paucity of our exact knowledge of the pathology of the fœtal and the infantile brain. Cotard and Gandard cite seven cases with the symptoms of Strümpell's so-called polio-encephalitis, in which the cortex was found to be normal, and in which the lesions were as follows: cyst in the corpus striatum, cyst in the frontal lobe beneath the cortex, sclerosis beneath one lateral ventricle, clot in the internal capsule, embolic softening of the corpus striatum and surrounding tissue, hemorrhage into the thalamus opticus and corpus striatum. Besides this, Hóven records a cyst of the internal capsule, and in this case an especially careful examination was made of the cortex, which was found to be perfectly normal. Wallenberg also reports a cyst beneath the anterior corpora quadrigemina, implicating the crus cerebri and the lemniscus, and destroying completely one red nucleus. The title polio-encephalitis is the more unfortunate, as the same name has been very properly given by Wernicke to a thoroughly authenticated disease of the motor nuclei of the medulla oblongata (see p. 296), which

are the true medullary analogues of the anterior cornua of the spinal cord.

In 1865 Virchow described what he termed congenital encephalitis, consisting of little yellowish masses in the white cerebral substance, and referable, he thought, to interstitial inflammation of the cerebral substance, the color coming from fatty granules. Although it has been maintained by Jastrowitz that fatty granules are physiological in fœtal brains, there yet seems reason to believe that Virchow's description applies to certain infrequent cases.

Heschl, in 1859, gave the name of porencephal (*πόρος*, "a hole;" and *ἐγκέφαλον*, the brain) to certain exceedingly curious losses of cerebral substance in fœtal and infantile brains, varying in size from small cavities to an entire absence of both hemispheres. These cavities are generally full of liquid, and are traversed by filaments forming light and incomplete partitions. Substances resembling adipocere are sometimes found floating in them, or other substances of transparent, citron, yellowish, or brown color. These cavities may open into the arachnoid cavity, although they generally have a vascular membranous cover, the external face of which may be colored orange, yellowish, or brownish. Ordinarily the pia is absent. Kundrat maintains that the convolutions bordering upon these cavities have a radiating form in the cases of pre-natal origin. The adjacent convolutions may be carpeted with a fine cerebral débris, or may undergo a gelatinous transformation. Sclerosis of the tissues bordering the cavity is very frequent, and these walls often have a rusty color, probably from hemorrhage. Near the lesion diminution or obstruction to an artery may be found. Instead of the cavities, however, there may be absolute loss of one hemisphere or both. The bones may be thinned or thickened, the skull may be hydrocephalic or microcephalic in shape, occasionally the front is very much flattened and slopes backward. Porencephaly is generally fœtal in origin, after this being most frequent in first infancy, occurring exceptionally afterward. In fifty-seven cases analyzed by Audry, thirty-four were probably of fœtal origin, thirteen in the first two years of life, nine in the second infancy—viz., three in the third year, one at three and one-third years, one at seven, one at nine and a half, one at eleven, one at fourteen—and one in an adult. These singular losses of cerebral substance have been attributed to an arrest of development, extreme hydrocephalus, embolism or hemorrhage, encephalitis, and a profound cerebral anæmia. Kundrat endeavored to trace a constant relationship between vascular lesions and the porencephalic areas, but indubitable cases have been reported demonstrating that this view was not tenable. A peculiar gelatinous and cellular infiltration seems, in certain cases, to be one of the early stages of the process. The predisposing and exciting causes are but little known, though traumatism would seem to have been a distinct etiological factor in several instances.

The apoplexy of the newborn is regarded by Osler as one of the chief causes of the bilateral hemiplegia or paraplegia occurring at birth, and there can be no doubt that it is a very frequent condition

in newborn children—especially, as Sarah J. McNutt has shown, in conjunction with abnormal labor, asphyxia, and convulsions.

Thrombosis of the cerebral veins is regarded as a frequent factor by Gowers.

It will thus be seen that the pathological behavior of the fœtal and infantile brain is different from that of the adult one. In the former we encounter much more frequently meningeal hemorrhages and acute forms of encephalitis and meningo-encephalitis, whilst the rapid losses of cerebral substance of porencephaly are practically unknown at later periods of life. Nor should we find it surprising that there is such easily effected retrogressive metamorphosis of the cerebral substance in the infant, when we consider that the intracranial contents are the last portions of the nervous substance to develop, that they are very imperfectly developed during fœtal and infantile life, and that their great relative bulk, badly protected by the imperfectly ossified cranium, renders them extremely liable to injury from without in the helpless fœtus and in the almost equally helpless infant. It is a singular fact, however, that most of the lesions are in the motor tract that extends from the motor convolutions to the muscles. The course of this motor tract (pp. 80 *et seq.*) is through the corona radiata to the internal capsule, thence through the pons to the region of the decussation, where it divides into two columns, the one going down upon the same side into the anterior pyramidal columns (columns of Türck), the other crossing to the opposite side to pass down through the lateral pyramidal column, thence into the anterior cornua, making direct connection with the great ganglion-cells, from which arise the motor nerves that emerge along the anterior roots to terminate in the motor end-plates of the muscle. Although different convolutions are not infrequently the site of lesion, even as far back as the occipital lobe, the motor convolutions are pre-eminently affected. But lesions have also been found in the intracranial portions of this motor tract without affection of the convolutions; thus, as has been already stated, one of Gandard's cases had a clot in the internal capsule, with softening and ecchymosis of the Sylvian fossa; another had a focus of softening in the region of the internal capsule; and another a softening of the corpus striatum and the surrounding region. One of Wallenberg's cases had a hemorrhage in the right optic thalamus and corpus striatum; and another a cyst beneath the anterior corpora quadrigemina, implicating the pes and lemniscus and destroying fibres from the third pair and red nucleus; whilst Hóven's case had a cyst in the nucleus caudatus. These lesions act more disastrously upon the fœtal and infantile brain than upon that of the adult, for the evolution of the former is seriously hampered. The condition of the spinal and peripheral portions of the motor tract has yet to be studied thoroughly, although in some cases of spastic paraplegia there is reason to believe that there is either a descending degeneration or a lack of development of the intraspinal motor strands. There can be no question that the motor tract is peculiarly liable to disease in children, and that the cerebral palsies are the earliest, in point of time, of the great series of lesions to which it is liable at this period of life.

Dividing the motor tract into three portions, the intracranial, the spinal, and the peripheral, we may classify its lesions in this manner: cerebral, the cerebral palsies; spinal, myelitis of the anterior cornua (poliomyelitis anterior); peripheral, pseudo-hypertrophic paralysis; whilst progressive muscular atrophy is sometimes of spinal and sometimes of peripheral origin. The spinal and peripheral portions of the motor tract, however, are much more prone to chronic disease in children, almost the only exception being the acute onset of most cases of poliomyelitis anterior, whilst the intracranial portions of the motor tract are very liable to acute disease, the chronic lesions being rare. The cellular processes of the spinal lesions have been admirably studied, so that one of the most certain facts in pathology to-day is the disappearance or injury of the ganglion-cells of the anterior horn in poliomyelitis and in the spinal forms of progressive muscular atrophy. It is not as yet quite certain whether the muscular wasting of the peripheral forms of progressive muscular atrophy and the commingled wasting and hypertrophy of the pseudo-hypertrophies are due primarily to the affection of the muscle-cells, or are secondary to changes in the anterior cornua that have not thus far been detected by the means of microscopical preparation at present in use, although the evidence is as yet entirely in favor of the former view. But we are sadly in lack of knowledge as to the exact pathological nature of the lesions in the intracranial portion of the motor tract. Porencephaly, as we have seen, would seem to be a cellular process independent of preceding arterial impairment, inasmuch as Kundrat's opinion, that the porencephalic loss of substance is always in an arterial range, has been disproved. The fœtal and the infantile brain must be subject, therefore, to sudden cellular disintegrations, such as we see in the anterior horn of the spinal cord in poliomyelitis anterior, although on a much larger scale, and such as are not seen in the adult brain except in the rare cases of idiopathic softening that have been described by Wernicke. Arterial and capillary hemorrhage and arterial and venous thrombosis are also important factors, as we have seen, but we do not know what relationship they bear to the atrophies, the sclerosis, porencephaly, or the cerebral porosity of Golgi.

Prognosis. The extent of the cerebral lesion, as manifested in the contractures, the paralyses of motion, the mental changes, and the exaggerated tendon-reflexes, will indicate the prognosis. A careful examination should therefore be made in each case to determine the amount of damage done, which will be ascertained by these symptoms. Tests of the motor and sensory power of the paralyzed limbs should be made. Examinations of the eye and ear are important. The tendon-reflexes should be tested and the amount of contracture ascertained. Mental tests should be applied, and the impairment of cerebral substance resulting from disease should not be confounded with the intellectual dulness occasioned by lack of mental training and the petting and spoiling usual in the case of a paralytic and helpless child.

Treatment. Very little can be accomplished by treatment in such cases. The faradic current is undoubtedly the most efficacious

agent in the cases of hemiplegia, especially those taken in the early stage, its use sometimes proving very beneficial indeed. The case represented in Figs. 149 and 150, for instance, has made an excellent recovery, as has been said. In paraplegia, however, it has proved quite useless in my hands, and in bilateral hemiplegia it has not been of marked benefit. Osler speaks of massage, with vigorous flexion and extension of the limbs, as having been of great service, but I have not found it of any special use except in the before-mentioned cases of recent hemiplegia. The knife and apparatus of the orthopædic surgeon are usually the only relief for the spastic defects. In proper cases, careful and special education may greatly help the mental defects. Surgical operation upon the brain is preposterous, both on account of its probable hopelessness in case the lesion can be localized, as well as on account of the extreme difficulty of such localization.

INTRACRANIAL GROWTHS.

ETIOLOGY. The causes are—

Sex;
Age;
Heredity;
Trauma;
Nervous strain.

Males are very much more subject to intracranial growths than females, in a proportion of about two-thirds of the former. Most of the cases occur between the twentieth and fortieth year, and more frequently after this period than before it. In children cases are rather more rare under eight than after.

Age seems to have somewhat of an influence upon the variety of growth; thus children are more prone to tubercle, young adults to glioma, and sarcoma and cancer occur after maturity. Gumma is pre-eminently a malady of adults.

Heredity does not play much of a part except in some cases of cancer.

Trauma is an occasional cause.

I have seen a number of cases of cerebral tumor develop after great nervous strain, such as business cares and domestic anxiety, and bearing such a relationship to these circumstances as to warrant me in considering them as true causes.

CLINICAL SYMPTOMS. In every case of an intracranial growth there are two sets of symptoms, namely, the general and the localizing. The former are due to the disturbance of the cerebrum or cerebellum as a whole, whilst the latter are from local implication of some portion of the cerebrum or cerebellum.

The general symptoms consist of—

Headache;
Vertigo;
Vomiting;
Optic neuritis;
Convulsions;
Mental disturbance.

Headache is one of the most constant symptoms. It may vary enormously in character, however. The older writers were prone to dwell upon the severity of the headache of intracranial growth, but this has not been borne out by experience. In one case of my own, for instance, where a round-cell sarcoma of the size of a hen's egg involved the centrum ovale, and in which the time from the onset of the first symptoms to death did not extend over two months, the headache was very slight, non-localized, and vague; in another case of a still larger tumor involving the temporo-sphenoidal lobe, in which the duration was some five months, the headache was a dull ache, with sharp exacerbations of a few hours' duration, over one brow; in another case of small cystic sarcoma of the size of a hickory-nut, just beneath the white matter of the left ascending parietal convolution, the duration of the symptoms being about three weeks, the headache was slight until the last week, when it merged so rapidly into coma that its characteristics could not be determined; and so I might multiply instances of the variability of this symptom. On the other hand, I have known of excruciating pain; but the rule is that the headache is not violent except in the cases of intracranial syphilitic growths, which, however, as we shall have occasion to see, are usually conjoined with a gummatous meningitis. In children intracranial growths are very frequently accompanied by no headache at all. I do not, therefore, think that the violence of the headache is of any diagnostic value in intracranial growths, although I do believe that in adults some form of headache, however slight and variable, can constantly be ascertained by searching inquiry. Nor does the location of the headache usually bear any relation to the site of the growth, as I have demonstrated over and over again to my own satisfaction. Indeed, with a history of fifty cases before me, I have never yet found the pain a localizing symptom, except that in twelve it was upon the same side as the lesion, but bore no exact relationship to it. Of course, if the trigeminal nerve at the base is affected, there may be pain limited to the area of distribution of this trunk; but this is a localizing symptom and not a general one—in other words, it is due to local implication, and not to disturbance of the cerebrum as a whole. Nor have I found that localized tenderness is of any more value diagnostically.

Vertigo as a general symptom—*i. e.*, due to the disturbance of the cerebrum as a whole—is generally slight and vague, no more than is seen in many other diseases of the brain, and indeed it is not nearly so apt to be so severe as in most cases of lithæmia. A distinction must be made here, however, between vertigo as a general symptom and vertigo as a localizing symptom, because when it affects the cerebellum, for instance, or the peduncles of this organ, it may become very pronounced in a characteristic way, which has been spoken of on page 149. I should therefore lay down as an axiom that pronounced vertigo without the special characteristics of cerebellar vertigo is not due to an intracranial growth, but is much more likely to be due to lithæmia, some lesion of the aural apparatus, perhaps of the temporal lobe, or some ocular strain.

In 568 cases tabulated by Jacobi vomiting was noted in only 172; in 40 cases collected by Knapp it was observed in only 17; but in 50 cases of my own it was observed 45 times, and I believe that, as in headache, a history of vomiting or nausea will be found in almost every case. Like headache, however, nausea and vomiting may be excessively variable, so that on the one hand it may be so persistent as seriously to endanger life, whilst on the other hand it may be so slight as to consist of nothing more than occasional qualmishness.

There has been an attempt made to create a distinction between optic neuritis and so-called choked disk; but this does not appear to be warranted, as in both cases there is a true neuritis of the optic-nerve fibres of the ordinary nature of degeneration. Great stress is laid upon this symptom by almost all writers, and, in my opinion, too much. According to the careful examination of cases made by Knapp, optic neuritis was present in over two-thirds of the cases examined. Gowers estimates the percentage as four-fifths of all the cases; Bernhardt found either atrophy or neuritis in 25 per cent., amblyopia in 20.4 per cent, and normal vision or normal fundus in 6.8 per cent., 47.8 per cent. of the cases giving no data; Oppenheim determined the presence of neuritis on one or both sides in 82 per cent. of his 23 cases; while Annuskee and Reich found neuritis absent in only 5 per cent. of 88 cases. I cannot agree with this estimate of the frequency of optic neuritis, at least for practical purposes, for in 20 of my 50 cases there was no neuritis found. It is true that in only 30 of these cases of my own were repeated examinations made of the retina throughout the course of the disease, and it is therefore possible that in the other patients such a neuritis might have been found; but for practical purposes my estimate is more apt to be the correct one, I believe, because but few of us can wait weeks or months to make a diagnosis, and the practical question therefore is as to how often an optic neuritis is to be found in the earlier stages when the diagnosis is obscure, and not as to how often it may be found when such cases are repeatedly examined through weeks and months of the duration of the malady, by which time probably the diagnosis can be made by other symptoms.

Convulsion, like headache and vertigo, may be both a general and a localizing symptom. When it is due to disturbance of the cerebrum or cerebellum as a whole it is generalized, but when it is due to implication of a particular portion of the motor tract it is limited to certain muscles, to one extremity, or one side of the body.

The mental disturbance in intracranial growth is usually slight, although exceptionally syphilomata (which are generally accompanied by gummatous meningitis, as has been said) may give rise to varying mental symptoms, such as mania, or even general paresis. As a rule, however, the mental symptoms consist of such slight changes of the mind as to be scouted by the patients and relatives, although they can usually be detected by the skilled observer, and consist of some slight impairment of judgment, some heaviness that can be dispersed by a conversation or attracting the patient's attention, by loss of

interest in the subjects which have before interested him, or by a tendency to somnolency or coma. Of these changes, I consider the tendency to somnolency as by all means the most important one; indeed, in any case of gradually supervening coma with a precedent history over weeks or months of vague cerebral disturbance, I should suspect an intracranial growth, after excluding nephritis, diabetes, and trauma. I should have a suspicion, too, amounting almost to a certainty, of intracranial growth in a case of recurring headache, either slight or severe, with recurring periods of somnolency. Variations in the pulse, respiration, and pupils occur occasionally in varying degree throughout the career of an intracranial growth, but none of them is of any special value as diagnostic of such a growth. Occasional attacks of pseudo-apoplexy may also be observed, either from the growth of the neoplasm or from hemorrhages around and about it; and, of course, signs of disturbance of the organism as a whole are usually present, such as emaciation, anæmia, digestive disturbance, anorexia, etc. And toward the last the usual signs of dissolution common to so many nervous troubles may appear, such as vesical and rectal paralysis, bedsores, irregular respiration, etc.

Localizing Symptoms. The localizing symptoms of intracranial growths will depend, as a matter of course, upon the particular portion of the cerebrum or cerebellum which is affected. (*Vide* Chapter I., "Localization of Lesions," etc.)

Pathology. Knapp, Starr, Bernhardt, and Birch-Hirschfeld have tabulated 1139 cases, and to these I can add 50 cases of my own, making 1189 cases in all. An analysis of these shows that of the different forms of intracranial growths tubercle is the most frequent, then sarcoma and glioma, and next in frequency are gummata, cancer, and parasitic cysts, whilst the remaining varieties are rare. The different forms of intracranial growths, therefore, are—

Tubercle;
Sarcoma;
Glioma;
Gumma;
Cancer;
Parasitic cysts;
Fibroma;
Osteoma;
Enchondroma;
Myxoma;
Lipoma;
Angioma;
Neuroma;
Adenoma;
Cholesteatoma;
Actinomyces;
Aneurism.

Tubercle is by far the most frequent form of tumor, and is the most common in children and young adults. As has been said in Chapter VIII., "Tubercular Meningitis," in which a fuller descrip-

tion will be found, there may be either tuberculous meningitis with tubercular nodules, or the latter may occur alone.

Sarcoma may develop from the dura, pia, or the brain-substance. It may be solitary or multiple, being flat in shape, or fungus-like, or wedge-shaped, or it may occur simultaneously with growths in other parts of the body, when, it is said, it is generally roundish in shape. Internally, the sarcoma may have hemorrhagic, caseous, cystic, or fatty formations. When the usual round or spindle-shaped cells are predominant it is known as a round-cell sarcoma. When cysts are present in the tumor it is known as a cysto-sarcoma; when the cells contain pigment it is a melano-sarcoma; when there is a structure in the neoplasm like that of a lymph-gland it is known as a lympho-sarcoma; and when the fibrous tissue is predominant it is a fibro-sarcoma. In my cases the most common form has been the round-cell sarcoma, next the melano-sarcoma, and then the cysto-sarcoma. Besides these forms which have been enumerated, there is a glio-sarcoma, or a mixture of the pathological elements of a glioma with the round- or spindle-shaped cells of a sarcoma; telangiectatic sarcoma, in which the growth is very vascular; the plexiform angio-sarcoma, in which the cells are clustered around an enlarged vascular plexus; and angiolithic sarcoma, in which there is calcareous transformation; but these varieties are rare. The round-celled sarcoma, the most common form, is often confounded with epithelioma if it arises from the dura and is of the so-called endothelioma type, when it is usually flat and may grow downward into the cerebrum or outward into the skull. Histologically the distinction is usually extremely difficult, and it is doubtful whether it can really be made.

Glioma is usually solitary, and is most frequent in the cerebrum, next in the cerebellum, and then in the pons and surrounding structures. It has a tendency to recur, although this is slight, and it may attain a considerable size. It develops from the neuroglia and connective tissue of the central nervous system, and a reference to "Syringomyelia," in Chapter IV., will show that it has certain peculiarities that are very apt to be overlooked. It is pre-eminently a tumor of the parenchyma, and not of the membranes. Outwardly it may not show any signs at all. It infiltrates the nervous substance, and there is no line of demarcation between it and the surrounding healthy tissue. It may produce softening or even cysts, but it does not seem in the intracranial contents to have the same marked tendency in this respect that it does in the spinal cord, probably because of the different conditions of environing pressure. The sub-varieties of glioma are the telangiectatic, where there is an excessive development of vessels; a fibro-glioma, when the fibrous tissue is greatly increased; a myxo-glioma, when mucous tissue develops within the growth; and a glio-sarcoma, when there is a large number of round or spindle-shaped cells.

Gumma is usually only one of the varied lesions of an intracranial syphilis, which consists of pseudo-meningitis, an infiltration of the nervous parenchyma, and occasional nodules or distinct outgrowths, which are called gummata. The solitary gumma is com-

paratively rare, and especially so in children. The pathology of these growths is treated of in the chapter upon "Intracranial Syphilis."

Intracranial cancer may be primary or secondary, usually the latter. When cancer arises primarily in the brain it does so from the epithelium of the choroid plexuses, or from the epithelium of the ependyma, and possibly from the hypophysis or in the transverse fissure. When it appears in the skull or in the brain-substance it is usually secondary to these primary growths of the epithelium or the choroid plexus and the ependyma, or from secondary growths outside the skull. These secondary growths are usually small and rounded when in the brain-substance, or when they perforate the skull they may form one of the fungi hæmatoides.

Parasitic cysts may be caused by echinococci or cysticerci. When they are due to the echinococci, they are the so-called hydatid cysts, may vary in size, and be single or multiple, usually lying upon the brain, causing softening. The cysts due to cysticerci are of the form of small fibres, with scolices (*i. e.*, one of the cystic worms) of the racemus form.

Fibroma is rare. It is a rounded growth, hard in consistence, and consists of a fibrous tissue with a few cells. It may, however, be intermingled with the texture of a sarcoma, constituting the fibro-sarcoma already mentioned, or there may be calcareous deposits constituting one of the psammoma. Fibrous thickenings are sometimes found in the epithelium or the ependyma, and are regarded as being organized thrombi.

Osteoma usually proceeds from the skull, although it may arise from the dura, the falx, or the tentorium, and it rarely develops within the nervous parenchyma. There may be pseudo-fibroma, or pseudo-sarcoma may occur.

Enchondroma arises from the bones of the skull or dura, and lies upon the brain. It is cartilaginous in formation. Pseudo-enchondroma may also occur.

Myxoma is a soft, round, moderately developed growth, and is rare, much more so than the mixed forms, consisting of the so-called myxo-sarcoma and myxo-glioma.

Lipoma is extremely rare.

Angioma is also rare, much more so than when it is combined with sarcoma and glioma in the so-called telangiectatic varieties.

Neuroma is also rare, and consists of two forms: the heterotopy of gray or white matter that has been described by Virchow occasionally as of congenital development, and the neuroma, which is similar to the neuromata elsewhere found.

Adenoma is rare.

Cholesteatoma is also rare.

Actinomyces usually proceed from actinomycosis of the face and neck through the occipital foramen, giving rise to a diffuse purulent meningitis of the pia, with a formation of nodules or even of abscess.

Aneurisms of the large arteries at the base of the brain constituting

the circle of Willis may be sufficient in size to give rise to the symptoms of a neoplasm, but this is rare.

DIAGNOSIS. The diagnosis of an intracranial tumor is usually not difficult except in the very earliest stages, but occasionally it may become one of the most vexed questions in the whole range of medicine. In every case where there is a suspicion a careful history should be obtained from some reliable person who has been well acquainted with the patient, and it should always be remembered that the defects of memory or the slight mental changes of a patient may render him or her a totally untrustworthy witness. Headache should be carefully inquired for, and care should be taken to distinguish a headache that may have existed throughout the whole of life, as in cases of migraine, from one that has gradually or suddenly supervened. Vomiting or nausea should be carefully searched for, and we should be careful not to be misled by the general tendency of the lay public to attribute every nausea or vomiting to so-called "biliousness." Vertigo is not usually a symptom of much importance in intracranial lesions, except when it has the peculiar characteristic impressed upon it by neoplasm of the cerebellum or its peduncles; nevertheless, careful inquiry should be made for this symptom, because it will have a corroborating value in conjunction with those of more importance. In every case careful and repeated ophthalmoscopic examination should be made, and by one who is a skilled ophthalmoscopist. Fortunately nowadays ophthalmologists are so numerous and so skilful that there are very few communities in which this examination cannot be faithfully made. If a patient should have a marked headache, with vomiting or nausea, and also a neuro-retinitis, the general symptoms will usually be sufficient to base upon them a diagnosis of an intracranial growth, when proper exclusion has been made of other conditions which might produce headache, vomiting, and neuro-retinitis. As a matter of fact, however, it is rarely that a diagnosis can be made upon these general symptoms alone, and it is usually only done by the commingling of certain of the general symptoms with some of the localizing ones, such as have been already detailed. The diagnosis should be from—

- Nephritis;
- Locomotor ataxia;
- Myelitis;
- Intracranial syphilis;
- Paretic dementia;
- Multiple sclerosis;
- Chorea;
- Lithæmic neurasthenia;
- Lead-poisoning;
- Amaurosis from tobacco.

In nephritis there may be persistent headache, even with somnolency, and the arterial changes of the disease may give rise to paralysis, which is usually, however, of the hemiplegic form. I do not believe that the optic neuritis of nephritis can be distinguished from the optic neuritis of intracranial growth except in the early stage of

great œdema, but not in the later period of beginning or complete atrophic changes in the retina. But a careful and repeated examination of the urine microscopically, chemically, and quantitatively, will invariably determine the matter, although care must be taken not to lay undue stress upon a few casts or a slight amount of albumin alone.

I have seen cases of locomotor ataxia in which there was persistent pain about the head, especially in the ramifications of the trigeminus, accompanied by optic-nerve atrophy, in which a mistake had been made in the diagnosis, but a careful examination of the case would soon make the matter plain, because of the presence of the characteristic ataxia, the absence of the knee-jerk, the peculiar pains, and sometimes the trophic changes in the bones.

Occasionally what are reported as cases of myelitis will be accompanied by an optic neuritis. Seguin has reported three cases of this kind, one of which I attended with him. Here, of course, the diagnosis can be easily made, because the paralysis is both sensory and motor, is bilateral, may be accompanied by vesical and rectal impairment, there is no headache, and no mental changes whatever are observed.

Intracranial syphilis is often confounded with intracranial growths, and the differential diagnosis may sometimes be impossible. It can be made readily in that form of syphilis described in Chapter VIII., and characterized by the quasi-periodical headache, with obstinate insomnia, both of these disappearing suddenly upon the supervention of paralysis or convulsion. In syphilis of the base, with implication of the cranial nerves, preceded by this characteristic headache, the diagnosis can usually be made. In any case with a syphilitic history, in which there is evidence pointing to a neoplasm, the diagnosis can be made, I think, if the characteristic headache and insomnia have formed part of the history. But in cases of intracranial syphilis that are diffuse in character, or in which the gumma is merely a part of the general syphilitic infiltration of nervous parenchyma and membranes, and in which there is not a distinct syphilitic history, it may be utterly impossible to make the diagnosis. This syphilitic history is, as has been said, usually very difficult to obtain from women who may be entirely unaware of having been specifically infected. In any case, however, of a tumor implicating the base, even when there has been no syphilitic history, I think that we are warranted in suspecting intracranial syphilis, and we should hold our diagnosis in reserve until a careful trial has been made of large doses of iodide of mercury, or both these drugs; although it must be remembered that the gumma may give rise to considerable development of connective tissue in and around the tumor, and this cannot be affected by either mercury or iodide, so that in all cases these medicines may not determine the diagnosis. Nevertheless, they will usually have immensely more effect up to a certain point in the syphilitic tumors than in non-syphilitic ones.

The symptoms of paretic dementia without doubt occur occasionally as the result of intracranial syphilis. Sometimes I am inclined to

think that these cases will give an earlier history than is typical of an intracranial neoplasm, but it is undoubtedly a sad truth that in many instances this cannot be made out, and then it is impossible to make the diagnosis. This has been shown by the researches of Morel-Lavallée and Bélières. On the other hand, it occasionally happens, though rarely, that non-syphilitic intracranial neoplasms will give rise to symptoms of general paresis, and Knapp has detailed a very interesting case of this kind. It is fortunate for us in the present condition of our knowledge that this is rare, as there are no means of diagnosis. I have myself, however, never seen such a case.

Certain cases of multiple sclerosis, when the sclerotic patch is sufficiently localized to cause localizing symptoms, may cause confusion to one who is not familiar with the types of nervous disease; but the presence of the tremor, the nystagmus, the stupidity that occurs when the sclerotic process implicates large portions of the cerebrum, picture a disease that is different from that of tumor, characterized as the latter is by headache, vomiting or nausea; and tumors are apt to be considerably more rapid in their course than is sclerosis.

Chorea of the Sydenham type presents no similarity whatever to intracranial tumor in its clinical symptoms, although in both there may be a neuro-retinitis; but chorea accompanied or preceded by paresis or convulsions might lead to confusion in some cases, although stripping the patient and detecting the tell-tale choreic movements will clear up the diagnosis. Huntington's chorea might also lead to a mistake, but to anyone who is aquainted with the symptoms of the disease the mere mention of the fact of such possible confusion should be all that is necessary.

In lithæmic neurasthenia (*vide* "Neurasthenia," Chapter IX.) the vertigo is frequently so intense and obstinate as to cause a suspicion of intracranial growth; but the absence of a true headache and the presence of a sense of fulness or pressure, the fact that the vertigo is more intense than in cases of brain-tumor, and that it has not the characteristics of the vertigo of cerebellar disease, together with the entire absence of any localizing symptoms, will make the diagnosis easy. Some observers claim that they have seen optic neuritis in cases of lithæmic neurasthenia, but I have never perceived it.

In lead-poisoning the diagnosis is easily made by means of the lead-line on the gums, the flexor paralyses, the history of an exposure to lead, etc.

Amaurosis from tobacco, caused by optic-nerve atrophy, might lead to a mistake, but the entire absence of the general or localizing symptoms of intracranial growth should make the diagnosis by exclusion.

Prognosis. The prognosis of intracranial growth is in the main unfavorable, although it may be considerably modified by three circumstances: first, the effect of treatment; second, the question of its being of syphilitic origin; and third, the feasibility of an operation.

As a rule, a fair idea of what the progress of the growth will be can be obtained by observing carefully what its progress has been, so that a tumor that has progressed very slowly will usually continue

to do so, and *vice versa.* Whether treatment materially affects the course of a non-syphilitic intracranial growth is a matter about which neurologists are in great doubt. Certain it is that under vigorous treatment some intracranial growths will not only be arrested, but will retrograde, and the patient may materially improve. I have seen this in several instances, and at the present time I number among my patients some six or seven who have remained in this quiescent condition for years, one especially in whom the growth has made no progress for nearly nine years after it had been seemingly arrested and caused to retrograde. While syphilitic tumors, so-called gummata, may attain to a stage of connective-tissue proliferation which cannot be affected by means of the anti-syphilitic remedies, yet in a number of instances vigorous treatment may cause a startling improvement; so much so that it is my custom to be very cautious about prognosis in such cases. I can recall several instances in which the patient was seemingly so near death as to be beyond reach of even surgical help, yet in which the symptoms have so markedly improved as to constitute a practical cure; *i. e.*, the patient is able to attend to his or her usual avocations, taking care, however, not to be subjected to great mental, emotional, or physical strain. More than this, it should be remembered that in all cases of gummata it is almost impossible to say how much of the symptomatology is due to the syphilitic infiltration of the cerebral or cerebellar parenchyma and its membranes, and how much is from actual gummatous formation; and, therefore, it is impossible to say as to what is the exact pathological nature of the cases in which there is cure. It is a common experience with me to see cases of intracranial syphilis with symptoms that are indicative of a lesion at the base and that have gone no further than some implication of the crura cerebri and the cranial nerves, particularly the third pair, become absolutely cured and remain so. The modifying effects of operation will be fully considered in the section upon treatment.

TREATMENT. In all cases of intracranial growth resort should be had to vigorous treatment by means of the iodide and mercury. This should be done even where there is no history of syphilis, both because the syphilis may have been overlooked, especially in the female, and because it is not yet positively known whether the iodides and mercurials may not modify the course of a non-syphilitic growth. Both in syphilitic and non-syphilitic growths I have much more faith in the iodide than in mercurials, for reasons that will be found fully stated in Chapter VIII., under "Syphilis of the Nervous System." The iodide should be pushed to the point of iodism, about which directions are given in the chapter just mentioned, and if mercury is used it should be made to produce ptyalism. In all cases in which, in spite of treatment, the growth is rapidly proceeding and inducing symptoms which may be fatal to life, such as severe convulsions and paralysis, or symptoms which may involve great danger to structure, as in rapidly advancing blindness, an operation should be done promptly if the tumor can be reached. But I am firmly convinced that we usually wait too long before

operating. There is nothing to gain by delay, except the bare possibility that a tumor may undergo a retrograde metamorphosis; whilst the danger is that of an intracranial growth. I believe, therefore, that when treatment has been accorded a fair trial an operation should be done immediately. Early operation should become a surgical axiom in these cases; and when it does, the rate of mortality will be materially lowered. Tumors of the cortex, of the centrum ovale, or of the cerebellum should be operated upon; but it is useless to attempt to reach them at the base unless they produce such symptoms as will probably localize them at the base of the frontal lobes, when there will be some impairment of the sense of smell.

There are certain inherent uncertainties, however, about an operation for the removal of an intracranial growth which should be carefully contemplated by the surgeon and explained to the patient or his friends. In the first place, while we may be reasonably certain in tuberculous or syphilitic tumors as to the real pathological nature of the lesion, we cannot be positive until the brain is opened how much of tubercular or syphilitic meningitis may accompany the gumma or tubercle, and therefore we cannot know until this time how much relief can be afforded. I have seen three cases of intracranial syphilis operated upon in which a gumma was found, but in which there was too extensive a meningitis to be removed by the surgeon's knife, all dying; but in two of these the tumors were found to be multiple, the largest one having produced the localizing symptoms that had dictated the operation. In gliomata, too, the infiltration of the cerebral or cerebellar parenchyma may be so widespread as to make it impossible to feel sure of eradicating it all with the surgeon's knife. My deceased friend, Dr. Frank W. Rockwell, operated for me many years ago upon a case of this kind in which death shortly followed, and in which it was ascertained at the autopsy that only a small portion of the gliomatous formation had been removed; and in another case operated upon by the same surgeon the operation was abandoned, after opening the skull, for this reason; very wisely, as the post-mortem examination showed. Then, too, it is impossible to tell beforehand as to whether a tumor is cortical or sub-cortical, and if it should prove to be the latter, it may be impossible to find it. Thus, in one case of mine to which I have elsewhere alluded (Fig. 22), a small cystic sarcoma lay just beneath the gray matter in the ascending parietal convolution at the junction of the upper and middle thirds, but it was not found at the operation; and even when the brain was taken out and dissected, it was detected with great difficulty. The reason was that it was a round-cell sarcoma, with a cystic degeneration in the centre that had given it a rather softer consistence than the surrounding cerebral substance. Although I thought I detected it by palpation at the operation, the surgeon would not agree with me, and at the autopsy it was found, marvellous to relate, that the probing-needle had gone through it in three different directions without giving us any sense of resistance to the touch. In another instance in the experience of an eminent neurological colleague, the tumor was not found at the operation, or at the autopsy,

or, indeed, until months afterward, when the brain had been hardened in bichromate salts, and careful dissections made of it, when it was discovered to be not larger than a pea. I know of several other instances of this kind. Another danger lies in the fact that the tumor may lie upon the same side as the paralysis of the limbs, not upon the opposite side, as generally happens; and this is due to the fact that in such a case only a small proportion of the motor strands have decussated and passed over to the opposite side as usual —a fact which was pointed out by Flechsig. Not long ago my friend, Dr. R. H. M. Dawbarn, operated upon a traumatic hemorrhage that I had diagnosed, selecting, as usual, the side oppo-

Fig 153.

Tumor of left temporo-sphenoidal lobe, causing hemianopsia, word-deafness, ataxic aphasia, hemiplegia, motor oculi paralysis, and convulsions.

site to the hemiplegia. Finding nothing upon that side, and the patient being too much exhausted to go on with the operation, it was abandoned, and the autopsy showed that the hemorrhage was upon the same side as the hemiplegia, whilst the cord showed but a partial decussation. Finally, the growth may be so large or so deeply seated as to render it impossible to remove it. In still other cases the lesions may be multiple, as in one case of mine where a melanoma of the angular gyrus caused hemianopsia, and another melanoma of the internal capsule caused hemiplegia, both sensory and motor; or, as in the case represented in Fig. 153, where a tumor caused word-

deafness, and also, by the pressure of the distorted cerebrum, ataxic aphasia, third-nerve paralysis, and hemiplegia; whilst another tumor, impinging upon the optic fibres as they entered the rear portion of the internal capsule (Fig. 39), caused hemianopsia. I believe, however, that the size or deep-seatedness of a growth should not stop us from operating on a patient who is certain to die unless relieved, because many cases of this kind have shown that in these days of antiseptic surgery a patient may thus be snatched from out of the shadow of death. Considering all these difficulties, therefore, the question arises as to what percentage of success we can expect in operations for cranial tumors. The accompanying table is based upon an investigation made for me by my friend, Dr. R. S. Newton:

Total number of operations for tumor to September 15, 1892.	Times failed to remove the growth when found.	Times tumor not found at point of operation.	Times trephined or operated for relief of pressure.	Recovered after removal of tumor.	Number of deaths.	Percentage of mortality.
110	5	15	7	63	42	$38\frac{2}{11}$

A perusal of this table will show that out of 110 operations 5 failed to remove the tumor. In 7 the trephining was only done to relieve pressure. Where, however, a growth has actually been removed, in 105 cases there were 63 recoveries and 42 deaths, the mortality having been $38\frac{2}{11}$. This result is to my mind exceedingly favorable for brain surgery when we consider that the first operation upon a brain tumor was done in 1884, only six years ago, because I have no doubt whatever that every year will witness great progress not only in our methods of localization, but also in our surgical technique. In any case, however, it should be remembered that, as was first suggested by Horsley in 1890, trephining for the relief of tumor may be of considerable therapeutic value in relieving pressure, even when the tumor is not found or cannot be removed. Some of the cases in the above table show a very remarkable success from an operation done with such an end in view—much too favorable, indeed, I think, to be warranted by the facts, as, in four cases of my own, death followed in every instance, although remarkable improvement was temporarily obtained in three of them. In one of these last-mentioned cases—the one represented in Fig. 153—indeed, the patient lived four months after the operation was so done, although he was at the point of death at the time, and considerable improvement was obtained, yet at the autopsy a tumor as large as a hen's egg was found in the depths of the temporo-sphenoidal lobe; and in another case of mine, operated upon three months ago, seemingly moribund at the time, there has been marked improvement.

Whenever the brain is trephined for the removal of an intracranial growth the former should be liberally exposed by making repeated openings with a trephine along a large arc, and then cutting with

a saw or *rongeur* forceps from hole to hole, elevating the bone and breaking its back upon the base of the arc in this manner. This should be done because it is impossible to diagnose minutely the exact site of the most pronounced lesion. A dental engine or electric motor is much the best instrument to operate with. When the brain is exposed the dura and the pia should be carefully dissected away, and, if the tumor is not found upon the surface, the nervous mass should be carefully palpated. If this does not locate the subcortical growth, a long, fine needle should be carefully inserted at different places and moved in different directions. This needle, however, should have the inserted end blunt and rounded, so that it will be more likely to give a feeling of resistance when it strikes a tumor. If this does not suffice, I can see no objection whatever to careful incisions, and I think that surgeons are very much more afraid of these than is at all warranted by the circumstances. It is only a question of time, after all, for the patient to die if the tumor is not removed, and a few incisions more or less can make no material difference, whilst a small tumor, such as a cystic sarcoma, which I have just spoken of, or a melanoma, would be detected in this manner. Horsley recommends that oozing from the diploë should be checked by plugging the bone with a wax made according to the following directions:

℞.—Ceræ flavæ . . . . 1 part.
Vaselini . . . . 4 parts.
Acidi carbolici 1 : 40 }
Ceræ albæ } . . . . . āā q. s. M.
S.—Boil before using.

But it does not seem to me that this is necessary, as such hemorrhage from the diploë can be much better controlled by compression with an artery-forceps for a few minutes. Some hyper-cautious operators object to opening the dura should nothing appear after removing the bone, but this seems to me rank nonsense in these days of antiseptic surgery. It is generally supposed that a subcortical growth will be disclosed by discoloration of the brain or a bulging, but this is seldom the case. All the bleeding vessels should, of course, be ligatured, and a 20 per cent. solution of cocaine may aid in controlling the troublesome hemorrhage; or, as Roswell Park has suggested, a 5 per cent. solution of antipyrin. Surgeons are divided in opinion as to the question of drainage, and Horsley has abandoned it; but I have never seen any ill results in my cases from it. If no tumor has been found, a liberal portion of the bone should not be put back; but if the tumor has been located, of course there is no objection to putting all the fragments into place, these having been kept warm and aseptic until such time. I presume, of course, that no surgeon would undertake an operation of this kind without the most strict regard to antisepsis. I have seen cases operated upon in both ways, and whilst I have never seen any sepsis result from an operation done antiseptically, in two cases in which antisepsis was disregarded there was not only the most frightful sepsis, but in one of them there was the most horrible purulent encephalitis that it has

ever been my fortune to meet with upon a post-mortem table. The further risks of the operation are shock and hemorrhage. The first of these will depend upon such well-known surgical considerations as the extent of the growth, the length of time consumed in removing it, and the physical condition of the patient; and is, of course, to be met by the ordinary means. Hemorrhage to a fatal extent is exceptional, although the German surgeon Von Bergmann lays considerable stress upon it. The convolutions can be reached in accordance with the rules that are formulated under the heading "Cranial Topography."

HYDROCEPHALUS.

Hydrocephalus may be either subdural or ventricular. The former variety is known as external hydrocephalus, whilst the latter is the internal. It may be either primary or secondary—*i. e.*, the result of other lesions of the nervous system; and it may be of an acute or chronic type.

Acute hydrocephalus is usually caused by tubercular meningitis, and the fluid may be either subdural or ventricular. The effusion is caused by the meningitis of the pia, the lining membrane of the ventricle, the choroid plexuses, and the velum interpositum. Occasionally, however, the hydrocephalus may be acute and febrile, and in these cases the choroid plexuses have undergone inflammatory alteration, there is a granular condition of the lining membrane of the ventricles, the surrounding brain-tissue is softened, and the brain substance is compressed, whilst the external meninges are apparently healthy.

Chronic hydrocephalus may be external or internal. The external form is usually a senile phenomenon, and it may occur in many conditions of wasting of the brain or arrested development. In other cases without any atrophy of brain-tissue the subdural fluid is in excess, when all that can be detected post mortem is the undue amount of fluid, without membranous alteration, the real causation being unknown. In this external form there is sometimes a sacculation caused by adhesions between the dura and pia, and this may occur over a hemisphere or beneath the tentorium, so that there is corresponding unilateral enlargement of the skull. These cystic formations are probably of inflammatory origin, and they may be accompanied by intercurrent meningitis. The external form of chronic hydrocephalus is congenital or acquired. The former is usually of fœtal development, and its etiology and pathology are not known, except that it sometimes seems to be hereditary. It may affect all the ventricles or the fourth ventricle alone. In this variety the shape of the skull is characteristic, and it becomes more marked after birth. The fontanelles are conspicuous and bulging, and the bones are often separated at the sutures. The cranium is generally disproportionate in size to the face and of a rounded shape, as in Fig. 154. The frontal skull projects, the orbital plates are oblique, and the eyes look downward and the lower lids overhang them. The duration of

this congenital hydrocephalus is variable, death usually occurring in a few months, although exceptionally the patient may live on to adult or old age, probably because the progress of the lesions is arrested and the fluid is absorbed, when ossification of the bones may be completed with the development of the so-called Wormian bones. The mental and motor symptoms are persistent throughout life in less degree, however. The acquired form of chronic internal hydrocephalus may be secondary, being usually caused by mechanical or meningeal lesion and the primary form. Obstruction of the venæ Galeni, tumors, abscess, and meningitis are lesions which

FIG. 154.

Case of hydrocephalus.

act by either hindering the return of blood from the vessels within the ventricles, or obstructing the passage of fluid out of the ventrcles through the openings in the membranes of the fourth ventricle, the foramen Magendie and Merzejewski's apertures on each side of the roots of the glosso-pharyngeal nerve. When these latter canals are blocked by a meningitis, the interchange of fluid between the ventricular systems becomes disarranged, and the effusion may be confined to the lateral ventricles. It must be said, however, that the pathology of this form has not been satisfactorily explained.

Hydrocephalus may occur without any of these lesions, and then its causation is utterly beyond our knowledge.

The symptoms are akin to those of tubercular meningitis of a low grade, but the distinction can usually be made by the fact that the clinical phenomena are vague and by no means so distinct as in true tubercle. They are extremely variable, consisting at times of mental defect, sometimes actual idiocy, whilst upon other occasions there is a considerable degree of intelligence. The child is usually unable to walk, not so much from lack of muscular strength as from ability

to co-ordinate. Convulsions, contractures, fever, and vomiting may occur. The head is not held erect, but lolls in different directions. The eyeballs are restless, the hair is scanty, and the skin of the head is thin. In some cases amaurosis may occur, when usually an atrophy of the optic nerve will be found. Temporal hemianopsia is sometimes observed. In the adult the enlargement of the head is less common.

The diagnosis is to be from rickets and thickening of the skull-bones.

In rickets the head is square, the fontanelles do not bulge, and the other signs of rickets are present.

Thickening of the cranial bones is easily distinguished from the fact that the progressive enlargement of the head is extremely slow, and the mind is generally unimpaired. I have seen one case in a man of fifty who was mentally far above mediocrity.

No diagnosis can be made between the external and internal forms.

The prognosis is always grave.

The treatment is practically *nil.* I have never yet seen a case relieved, and the temporary improvement has been of slight amount. The lateral ventricle can be punctured by the surgeon according to the rules laid down on pages 97 *et seq.*; but this method is sometimes fatal in results, and has never been of much use. In any case where some relief was absolutely insisted upon, I should much prefer to have a portion of the bone, two or three inches across and three or four inches long, removed over one side of the head when the hydrocephalus is unilateral, or over both hemispheres in a bilateral effusion. This bone should not be replaced. Puncture of the spinal membrane has been resorted to, and although it is harmless, it is also valueless. Compression of the skull by diachylon-plaster according to the method of Trousseau has been no more successful at my hands than the punctures. If it is desired to resort to this, the plaster should be one-third of an inch broad and applied in strips from each mastoid process to the outer part of the opposite organ from the hair at the back of the neck to the root of the nose; over the whole head so that the strips shall cross at the vertex; a strip three times around the head, first above the ears and eyebrows, and a little below the occipital protuberance. This compression, though, should be carefully watched. A broad elastic band, however, will exercise quite as much compression, it is claimed.

HUNTINGTON'S CHOREA.

Synonyms: Hereditary chorea. La chorée chronique. La chorée vulgaire chez les veillards. La chorée de l'adulte. Chorée clinique héréditaire. Corea ereditaria. Chorea chronica progressiva.

HISTORY. The first description of this disease was given by Huntington, a physician of Long Island, and his name has very properly been given to it. We are not as yet sufficiently informed as to the pathology to distinguish it by any better title. As chronic chorea was but little known previous to Huntington's paper,

I claim for my own country the merit that belongs to it for this discovery. Waters, of Franklin, New York, had remarked the same disease as far back as 1841 in a letter to Dunglison, and in 1863 Lyon had also observed it; but nevertheless the best description was that of Huntington, and he was really the first to portray accurately the malady. Since that time a number of cases have been observed by different authors—Landouzy, Maclaren, King, Russell, Sinkler, Ewald, Peretti, Déjérine, Lanois, Huber, and many others in different countries.

ETIOLOGY. The etiological factors having a certain relationship to Huntington's chorea are:

Heredity;
Age;
Sex;
Pregnancy;
Rheumatism;
Emotions;
Some functional nervous diseases, as hysteria, epilepsy, etc.

Heredity is a very constant factor in Huntington's chorea. There are many communities in different parts of America, to my knowledge, in which this form of chorea runs in certain families, and inasmuch as there is a great superstition in regard to it, it is very difficult to obtain much information about it. I can name at the present moment at least a dozen towns within easy reach of New York in which it is very prevalent, and yet in which it is nursed as a dark secret. This heredity has been well established in all the cases which have been published in other countries.

The hereditary progression is apt to be capricious at times, as it is in other hereditary maladies. Thus, in King's case, the great-grandfather was choreic. He had ten children, only four of whom had this chorea; concerning only three of these four are there very definite details, and it is only known that they had children who were also choreic; the fourth one of these four had nine children, eight of whom were healthy, but the ninth was choreic; this ninth one had five children, of whom four were choreic. Of these four, three had no children; but the fourth one had a chorea whilst he was still young himself, and was cured of it, when again at thirty-five he passed gradually into the typical Huntington's chorea.

Huntington's chorea generally commences in adolescence, attaining its maximum at adult age, contrary to the chorea of Sydenham, which, as we have seen, is found in infants, at which age Huntington's chorea is rare. The following table of Huet's embraces Huntington's observations and his own:

10 years	1	45 to 50 years	7
10 to 15 years	2	50 " 55 "	8
20 " 25 "	1	60 " 65 "	1
25 " 30 "	5	65 " 70 "	1
30 " 35 "	16		—
35 " 40 "	13	Total	66
40 " 45 "	11		

A congenital case is represented in Fig. 154.

From this it will be seen that most cases occur between the thir-

tieth and forty-fifth years, and this has been confirmed by tabulated statements of other authors. Even where there is no heredity, the onset at different ages is the same.

The female sex are much more prone to Huntington's chorea than the male, just as is observed in the chorea of Sydenham.

Pregnancy is a frequent cause of Huntington's chorea. Primiparæ are most affected (and during the first five months), although multiparæ may be affected who have never had the disease before.

Rheumatism is an infrequent cause in Huntington's chorea, although it may have been observed in the hereditary history.

The emotions are frequent causes.

The relations of Huntington's chorea to other nervous affections have not been thoroughly studied, but so far, hysteria in combination with it as a personal or hereditary antecedent is rare. Epilepsy is more frequently seen; migraine is extremely infrequent.

Clinical History. The onset is generally very gradual and progressive, but it may be sudden, and in some cases, after severe

Fig. 155.

Case of congenital Huntington's chorea.

Fig. 156.

A case of Huntington's chorea. One attitude.

relapses of ordinary Sydenham's chorea, the Huntington chorea may supervene. The muscular movements are entirely different from those of Sydenham's chorea. In the former, as pointed out in Chap. IX., under "Chorea," the muscular movements consist of fibrillary contractions that are jerky, beginning quickly and ending

quickly, or others that are not abrupt enough to be called jerky, and may rather be designated as wavy, as in the athetoid variety. In Huntington's chorea, however, the muscular movements are of far larger range than the fibrillary ones of Sydenham's chorea, and whole groups of muscles are set into action as in a voluntary movement, so that

FIG. 157.

A case of Huntington's chorea. One attitude.

FIG. 158.

A case of Huntington's chorea. One attitude.

the patient seems to be posturing and grimacing almost from eccentricity, and he has a dancing movement, with many queer contortions of the face and head, the whole picture at first exciting ridicule. (Figs. 155–158.) Most of the muscles of the body are generally affected, and the disease is never a localized one. In some cases,

however, the fibrillary movements of the Sydenham type are intermingled in slight degree with the dancing and grimacing ones. The muscles that are first affected are those of the face, especially of the mouth, and after these the limb, trunk, and other muscles become affected, although sometimes all are involved simultaneously. The speech is frequently altered in consequence of the muscular movements of the buccal cavity, pharynx, and respiratory diaphragmatic muscles. This alteration imparts to the speech a nasal, confused, obstructed, and sticky articulation. The external muscles of the eye are not often affected. Although most of the muscles of the body are attacked, nevertheless individual cases vary as to the muscles implicated, so that in some it is the extremities, in others the face, respiration, and deglutition; whilst in others again there is a hemiplegic distribution, or the lower extremities are implicated. There is seldom any impairment of sensation, and when there is, it is only some slight dulness of perception of painful sensations, which is probably due to the mental enfeeblement. The special senses are not altered. There is usually marked inco-ordination, evidently dependent upon the movements, so that the patient brings the forefinger to the nose in a series of zigzags and fails to hit the mark, or cannot walk a straight line, etc. There is no alteration in the response of nerves and muscles to either the galvanic or faradic currents. The sympathetic nervous system is not affected, nor the bladder or rectum, except in a very advanced stage. The urine is unaltered. There is almost invariably some mental impairment, generally consisting of slight and progressive dementia, which may pass on to absolute dementia. Memory is greatly enfeebled. At the onset there is apt to be a great tendency to depression, although this is not a true melancholia, but rather a depression of spirits, partly due to the disease itself—more largely, I think, to the fact that this disease is known to be hereditary and is looked upon with extreme dread in families who are subject to it. Suicide occasionally occurs, mainly from a well-founded dread of the consequences with which the patient is apt to be familiar. As the disease progresses, however, this suicidal tendency is apt to disappear. At times the patient is very irritable and subject to violent bursts of passion. In others there are vague ideas of persecution, and in others again there may be violent and dangerous outbreaks of mania. Occasionally there are hallucinations, especially of sight. As the disease progresses through many years, the patient may become so helpless as to be confined to the room or even to bed, and the dementia may become absolute, although many reach a fatal stage with but slight dementia and with but slight impairment of memory, and in some cases intelligence is not at all affected. These mental troubles usually supervene long after the motor troubles have been in existence, but occasionally the two sets of symptoms are synchronous. The duration of the malady is very chronic, and patients frequently live to be sixty, seventy, or eighty years, and even longer.

Pathology. The pathology of Huntington's chorea, like that of Sydenham's chorea and athetosis, is somewhat obscure. In one

case of Huber's there was found to be a cerebral pachymeningitis. In one of Macleod's there was a cerebral pachymeningitis and a hæmatoma of the left hemisphere, the latter extending from the posterior portion of the frontal lobe into most of the parietal lobe; the superior parietal convolution, the ascending frontal and parietal convolutions in their upper part, and some other of the parietal convolutions being thickened and atrophied. In another case of Macleod's several tumors of the dura mater were found in the left hemisphere implicating the first and second frontal convolutions, the upper portion of the ascending frontal and the ascending parietal, causing a depression and thinning of the cerebral parenchyma, but not leading to complete destruction of the gray matter. The arteries of the cerebrum were also atheromatous, and the cerebrum itself was firm and pale. The spinal cord was normal. In still another case of Macleod's there was a pachymeningitis and hæmatoma of each hemisphere, implicating the parietal lobes and the posterior portion of the frontal lobes, and reaching down to the fissure of Sylvius. The pia mater was congested and adherent. The meningeal vessels were dilated and varicose, and the larger vessels were atheromatous. The Pacchionian bodies were numerous and voluminous, particularly on the left. The cerebrum was abnormally small, and its convolutions generally atrophied, particularly of the superior parietal lobe. The cerebral substance was firm and congested, and the perivascular spaces very marked. But little fluid was found in the ventricles. The choroid plexus contained small gliomatous bodies. The walls of the lateral ventricles were red and granular, and the floor of the fourth ventricle was covered with large and thick granulations. The spinal membranes were congested, but the spinal parenchyma was sound. In Maclaren's case the dura mater and the pia were thickened, the Pacchionian bodies voluminous; and there was subarachnoid hemorrhage, covering the middle lobe on the two sides, but most on the left. There was general atrophy of the convolutions, abundant fluid in the ventricles, the cerebral substance was anæmic, a focus of softening was found in the centre of the right optic thalamus. The meningeal vessels and the anterior columns of the spinal cord were congested. In Berkeley's case there were dilatation and thickening of the arterioles, small necrobiotic foci around numerous vessels, amyloid corpuscles in the lymphatic sheath, vacuoles of the cord, and white substance, varicosities of the sheath of Schwann, absence of axis-cylinders of the nerve-fibres, and complete pigmentary degeneration of a large number of cortical cells with loss of their processes. Cellular alterations in the spinal cord were found, the cellular degeneration being especially marked in the spinal enlargements, whilst there was an antero-lateral sclerosis. The heart was affected, but without any case of old or recent endocarditis. In Bacon's case there was a chronic arachnitis, the cerebrum was atrophied, and there was much fluid in the ventricles. In Vassitch's case no lesion whatever was found in the cerebrum or cord. In Pan's case the cerebral meninges were found to be pale and anæmic, the cerebro-spinal fluid was abnormally large, the cere-

bral parenchyma was pale, markedly atrophied, the white and gray substance being somewhat less dense than usual, whilst there was softening of the spinal cord from the pyramids to the inferior third of the dorsal region, so that the medullary substance of the anterior half was converted into a diffluent pulp, whilst the posterior half was abnormally dense and resistant. In Charcot's case the cerebral meninges were found to be very adherent in certain places, particularly in the anterior and middle lobes, and where these adherences occurred the cortical substance presented a deeply rosy coloration and was resistant to a stream of water. The convolutions were very small, particularly the anterior ones, and there was such a large quantity of fluid in the meninges as to warrant the term œdema of the pia mater. In Tissier's case the cerebral pia mater was thickened, slightly congested, and adherent in only a few places. There were numerous foci of softening upon the external face of the two occipital lobes and the third and second frontal convolutions of the left hemisphere. This softening was entirely cortical, and absolutely no lesions of the heart or vessels were observed. From this *résumé* it will be seen that there is nothing about the pathological lesions to constitute anything pathognomonic of Huntington's chorea, although it will be noted that there has been a disposition to affect the motor convolutions and sublying strands and the motor portions of the basal ganglia. The lesions seem to be grosser than is the case in ordinary Sydenham's chorea, and constitute a marked type of meningo-encephalitis. The relative frequency of meningitis, hæmatoma, and gross arterial lesions, such as atheroma, is remarkable when compared with the infrequency of these in Sydenham's chorea.

Prognosis. The prognosis of Huntington's chorea is exceedingly grave, not only because a case has never been cured, but also because of the tendency to mental impairment and to the heredity being transmitted.

Diagnosis. The diagnosis of Huntington's chorea is made very readily. The characteristic dancing and posturing movements and grimaces, the mental impairment, the peculiar inco-ordination, oftentimes the progressive course of the disease and the heredity, all together make a picture that bears only a faint resemblance to any other disease. The maladies from which it should be differentiated are—

Sydenham's chorea;
Athetosis;
Locomotor ataxia;
Friedreich's disease;
Paralysis agitans;
Disseminated sclerosis;
Hysterical chorea;
Palmus.

Sydenham's chorea is a disease of infancy, whilst Huntington's chorea is a disease seldom occurring under the age of fifteen, generally between thirty and forty-five years of age, and it may therefore be practically called a disease of adult life. In Sydenham's chorea,

too, the movements are fibrillary, except in some temporarily violent cases, and these fibrillary movements, as I have again and again said, are jerky, beginning quickly and ending quickly, or the jerkiness may be merged into a gradual, wavy movement, as in the athetoid variety.

The movement of athetosis is wavy and gradual, usually of the hand. Double athetosis may cause a certain difficulty of diagnosis at times. It usually dates back to infancy, however. These movements are not only more gradual and wavy than the dancing movements of Huntington's chorea, but they are accompanied by rigidity and contracture of the affected parts, usually leading to marked muscular deformities, whilst neither rigidity nor contracture is observed in Huntington's chorea. The mode of progression is also different. The patient with Huntington's chorea dances and postures and grimaces as he goes, with a suppleness that is very suggestive of affectation. The double athetoids, on the contrary, are spasmodic and stiff in their movements, walking on the point of the foot, the knees approach so that they are knock-kneed, the arms are spasmodically held against the trunk, whilst the forearms are spread out as if balancing, and the fingers are affected with the peculiar stiff, wavy movement, at the same time that the trunk is held stiffly with the shoulders thrown back, and the speech, instead of being thick and explosive, is jerky, high-pitched, scanning, somewhat nasal and guttural.

Locomotor ataxia may have an inco-ordination that seems to an inexperienced eye to resemble the inco-ordination of Huntington's chorea, but in reality it is very different. The case of locomotor ataxia has no choreic movements of the dancing, grimacing type, and the inco-ordination is therefore a pure inco-ordination, not due to the choreic movements. In the gait of locomotor ataxia the heel is brought down with a flapping movement. In locomotor ataxia, too, mental impairment is not usual, and there is usually very marked sensory impairment of the sense of touch, muscular sense, and pain, and the bladder and rectum are often affected at an early stage.

In Friedreich's disease the diagnosis sometimes needs care. The movements generally affect one extremity or a segment of an extremity, and consist of a sort of instability of position, and not of the dancing movements of Huntington's chorea, for the former small muscular movements are not so multiple or various or irregular as in the latter disease. The motor inco-ordination is especially prominent in intentional movements, whilst in Huntington's chorea the choreic movements diminish with a volitional movement. In Friedreich's disease the patient grasps an object in a different way from what he would do if afflicted with Huntington's chorea ; for in the former the open, flat hand poises, as it were, for some time over the object before grasping it. In Friedreich's disease the gait is aggravated by closing the eyes. (Romberg's symptom.)

In paralysis agitans the characteristic visage and attitude (*vide* Chapter XIII., under "Paralysis Agitans"), the tremor that is so entirely different from the dancing movements of Huntington's

chorea, the slow, wise, deliberate, monotonous speech, easily enable us to make the diagnosis.

In disseminated sclerosis the tremor is a tremor, and not a dancing, seemingly voluntary movement, as in Huntington's chorea, whilst the speech is scanning, slow, jerky, and entirely different from the nasal, confused, sticky pronunciation of Huntington's chorea.

In hysterical chorea—chorea major or rhythmic chorea—as it is sometimes called, the diagnosis is easily made. This chorea manifests itself in single attacks, as do hysterical convulsions. The movements are rhythmical and of great variety, so that they may sometimes be dancing, hammering, swimming, etc. The attacks are provoked by compression or excitation of hysterogenic points, and are sometimes arrested by the same method, and there may exist such accompanying symptoms of hysteria as limitation of the field of vision, anæsthesia, color-blindness, etc. The picture is therefore a radically different one from the chronic, progressive Huntington's chorea, with its persistently dancing movements.

When palmus, or convulsive tic, is localized the diagnosis is easy; but when it has extended and become generalized the diagnosis may need some care, especially as palmus may be hereditary. The movements, however, are entirely different from those of Huntington's chorea. They are very brusque, rapid, instantaneous. They have not the waviness or dancing aspect of Huntington's chorea. The movements are automatic in that the same movements or order of movements are repeated again and again, and in this respect they differ markedly from the great variety and semi-involuntariness of the movements of Huntington's chorea. They are not continuous, but occur in paroxysms varying in duration. They can be arrested by a voluntary effort for a longer or shorter time, but this effort of suppression of them is generally accompanied by a feeling of extreme anguish, after which there is a sort of discharge, as it were, and the movements become more pronounced. By means of this voluntary control patients affected with palmus are often able to accomplish intentional acts during which the movements are completely suspended, whilst in Huntington's chorea there is only a diminution of the movement in performing the voluntary act, or in some cases suspension for a few seconds of the movements of the muscles that are concerned in the voluntary action, whilst the other muscles not concerned in this voluntary action are in motion with the characteristic movements. Palmus generally begins at a very early age, in childhood, or early adolescence, while Huntington's chorea is exceptional at this period, and is a disease of adults. In palmus, too, there often is ejaculation or exclamation of entire words, while in Huntington's chorea the sounds are inarticulate and are usually produced as in Sydenham's chorea by involuntary movements, such as movements of the tongue against the buccal cavities, the laryngeal squeaks, etc. In palmus, also, there is sometimes observed what is known as coprolalie —*i. e.*, an involuntary repetition of certain gross words or expressions; or echolalie, or echokinesia—*i. e.*, involuntary repetition of certain words which a patient is pronouncing, or of the gestures

which are made before him. These phenomena are never observed in Huntington's chorea. The mental alterations in palmus when present consist of fixed ideas, of certain emotional fear of places, the folly of doubt, and of touching, etc., whilst in Huntington's chorea there is a very gradual dementia with slight depression, sometimes at the onset, and there are slight losses of memory during the different stages of the malady.

TREATMENT. The treatment of Huntington's chorea is a matter of considerable doubt. I am inclined to think that if the malady were properly treated at the start it could be controlled; but the great dread of it, especially in the hereditary cases, causes the patients generally to be hidden from observation and sight. Certainly in the advanced cases which I have seen and treated the ordinary medication of chorea is absolutely without effect. I have tried arsenic, bromide, hyoscyamine, even hyoscine slightly, and have never yet been able to observe any result. I have never been able to try the electric currents, but I doubt very much whether they would have much effect, especially as the European observers claim to have seen none. Were I to have a case brought to me at the onset, I should certainly put it to rest and treat it with arsenic, Fowler's solution, commencing with two drops three times a day, running up to six or eight drops, with bromide of potash, ten to thirty grains in the day; and then, if this did not succeed, I should try hyoscyamine or hyoscine, $\frac{1}{100}$ grain once or twice a day, and apply the galvanic current carefully to the spinal column. I should, however, mainly rely upon the rest. But it must be borne in mind that this is pure speculation, as I have never been able to treat a case in this early stage myself, nor have I known of anybody else who has.

ATHETOSIS.[1]

Athetosis was first described by William A. Hammond, in 1871, and since that time the disease has been described by a number of other authors. He gave it the name because it was mainly characterized by an inability to retain the fingers and toes in any position in which they might be placed, as well as by their continual motion. Fig. 159 shows the hands of a patient afflicted with this disease. In Hammond's case the affection was unilateral. In 1872 Clifford-Albutt published the first case of double athetosis—*i. e.*, of athetosis implicating all four extremities. The movements of the fingers and toes are slow, wavy, and gradual, the fingers having a tendency to separate and the hands having a tendency to pronate, whilst the toes simply have a less tendency to separate, and none to flex or extend; and these movements are continuous, so that, as Hammond says, there is no fixed position. In some rare cases that have been described by Kinnicutt, athetosis has neither paralytic nor sensory troubles, although it is usually marked by some degree of imbecility (Fig. 159); but in the vast majority of instances it

[1] From ἄθετος, without fixed position.

is conjoined with contracture or paralysis of the affected limb, sometimes with muscular atrophy and hypertrophy, and in many cases there are marked symptoms of intracranial lesions, such as convulsive and apoplectic attacks, dementia, and cranial deformities. The so-called post-hemiplegic chorea is, in my opinion, nothing more than athetosis.

FIG. 159.

Case of athetosis.

Athetosis is a hereditary disease in a large proportion of cases, although the heredity consists rather of a family neurosis than especially of athetosis. It may occur at any time from infancy to advanced age. Sex does not appear to be of much etiological consequence, but prolonged or difficult labor, infectious maladies, traumatism, and other general causes have seemed at times to be causative factors.

Athetosis may occur from intracranial and spinal lesions, peripheral neuritis, and even from hysteria. The intracranial lesions are not fully understood, and the autopsies have been rare. The few autopsies have been made by Hammond, Bernhardt, Duchenne, Bourneville, Putnam, and others. There have been found pachymeningitis, cerebral and cerebellar asymmetery and atrophy, a slight degree of disseminated sclerosis, adhesions of the lips of the fissure of Sylvius to the frontal lobes, whilst Putnam could detect absolutely no cranial lesion visible to the naked eye, and Bourneville has had a similar case. Ross, Rolla, and Massalongo have found a secondary degeneration of the pyramidal columns of the cord, but Curella and Putnam had nothing of this kind in their cases, whilst Bourneville discovered a gray discoloration of the internal portion of the left anterior pyramid. In a case reported by Ringer there was a cyst in the posterior half or two-thirds of the lenticular nucleus, the cyst lying

in front, immediately without the inner capsule, and passing obliquely through the lenticular nucleus. It also involved the whole of the white matter lying outside and rather beneath the optic thalamus for about a half-inch. G. M. Hammond has also reported a case with tumor of the basal ganglia. The caudate nucleus was also involved, and a few of the fibres passing between the two nuclei of the corpus striatum were destroyed. Dr. Hammond is inclined to think that this is the only real description of the pathology that has been published, and that, as he stated in his original article, the seat of the affection was in the optic thalamus or corpus striatum. Landouzy and Sturges found respectively lesions of the lenticular nucleus and the corpus striatum; but the other lesions which have been already detailed and those which are about to be described gainsay this view. I believe that the cerebral lesions associated with cerebral palsies of childhood are often causative of this malady (*vide* "Cerebral Palsies of Childhood"). Athetosis has been observed in conjunction with locomotor ataxia, always of the bilateral or double form. It is also occasionally associated with neuritis, as in Löwenfeld's case, and with hysteria. Outside of these pathological findings, there is no doubt in my mind that athetosis is, more frequently than is generally imagined, a functional disease in the sense in which we apply the term "functional" to chorea; for, as stated in Chapter IX., under "Chorea," there are certain cases in which the movements are athetoid, and other cases of chorea in which athetoid movements are commingled with what I believe to be the true choreic movements, and the curability of these former is the same, so far as I have been able to perceive, as that of the latter.

The prognosis of athetosis, when it is organic, is very unfavorable, for no case has been either cured or ameliorated; but, as I have just said, the prognosis of the functional cases that are generally mistaken for those of chorea of the Sydenham type is the same as that of the chorea itself.

The differential diagnosis of athetosis is exceedingly easy, because the movements are not unlike those of any other form of nervous disturbance. Even in the athetoid chorea the waviness and gradual worm-like character of the movements are readily distinguished from the quickly beginning and quickly ending fibrillary jerk of the true Sydenham chorea. The so-called cases of post-hemiplegic chorea are really, as Dr. Hammond agrees with me in believing, cases of post-hemiplegic athetosis.

The treatment of athetosis, when it is of the organic type, is not known, for as yet no drug has been found to be of any value. It is probable, however, that the future will show that the athetosis which is conjoined with peripheral neuritis will be relieved by the same treatment as that of the neuritis. In athetoid chorea the treatment should be the same as that of Sydenham's chorea.

CHAPTER VIII.

DISEASES OF THE CEREBRO-SPINAL NERVOUS SYSTEM AND ITS MEMBRANES.

HYPERÆMIA AND ANÆMIA OF THE BRAIN AND CORD.

HYPERÆMIA and anæmia of the brain and cord are terms that are very glibly employed by the general practitioner to explain many symptoms whose pathology is obscure to him. But their use by the neurologist has steadily decreased within the last ten or fifteen years, until nowadays it is exceedingly rare to hear a diagnosis of this kind made in the neurological clinics; certainly in most of those of New York it is never made. Indeed, when, in my Inaugural Address as President of the New York Neurological Society, at a meeting held in May, 1890, I expressed myself as I shall do in this chapter, there was unanimous agreement with my views, with the exception of Dr. William A. Hammond, whom I had invited to be present. Most of the neurological text-books of late years have either omitted any chapter with these headings, or else have been very cautious in their statements.

The symptoms alleged to be those of cerebral hyperæmia or anæmia are headache, flushing or pallor of the face, delirium, vertigo, motor or sensory paralysis, retinal changes, slight aural symptoms, insomnia, and myosis. If these symptoms are examined one by one, the significance of each will become decidedly problematical to anyone who has had much acquaintance with the clinical aspects of cerebral disease. Headache, for example, is a very general symptom, indicating too many disorders to be of any service except in conjunction with other signs, and the statement that it is apt to be localized and widespread in cerebral hyperæmia is a pure assumption, of which no proof has ever been adduced. The condition of the capillary circulation of the face is by no means a reliable guide to the condition of the cerebral capillary circulation. Any hospital surgeon knows that marked meningeal hyperæmia is often associated with great facial pallor in fractures of the skull and the attending cerebral hyperæmia, just as great pallor may exist with many other hyperæmic intracranial conditions, whilst precisely the same pathological states may cause vascular flushing in other individuals. Delirium, like headache, is a general symptom indicative of too many conditions to be of any value by itself. The same remark is true of vertigo. Of motor and sensory paralyses as symptoms of cerebral hyperæmia and anæmia, it may simply be said that there is not an authentic case on record in which a simple anæmia or hyperæmia has caused a motor

or sensory paralysis. It is almost, if not absolutely, impossible to distinguish from the normal retinal changes consisting only of slight differences in vascularity, as every ophthalmologist knows, while the well-marked changes of neuro-retinitis have never yet been shown to have been produced by cerebral anæmia or hyperæmia. Tinnitus aurium and insomnia, like headache, delirium, and vertigo, are symptoms that are absolutely worthless by themselves. Neuritis is said to be a symptom of cerebral hyperæmia. It may be, but nobody has yet made public the proof. It will thus be seen that, of all these symptoms, headache, delirium, vertigo, tinnitus aurium, and insomnia are general symptoms which only derive a diagnostic value from their associates, while flushing or pallor of the face is an extremely doubtful indication of the intracranial circulation; motor and sensory paralysis and retinal changes are absolutely worthless as signs, and the diagnostic value of myosis is a pure assumption. Nor, when we take these symptoms together in a group, can we say that they are indicative of cerebral hyperæmia, because they are found in so many other diseases of the brain. Hammond affirms that cerebral hyperæmia can produce the symptoms of apoplexy, paralysis, convulsions, a soporific condition, mania, and aphasia. But when we turn to his chapter on the pathology of this particular form of cerebral disease we find the statements that the capillaries and large bloodvessels of the brain and pia mater will be found to be increased in size; that the white matter of the brain is increased in consistence and density, the gray matter is red or even violet in hue; there is a large amount of subarachnoid effusion, or even an effusion into the ventricles, and the choroid plexuses are often enlarged; if there have been repeated attacks of cerebral congestion, granules of hæmatin will be found in contact with the bloodvessels, or the latter may be unusually tortuous and have minute aneurisms. Now, I submit, this description is not that of a mere hyperæmia, but it is a condition of actual structural disease of the cerebral tissue, quite as pronounced as can be found in many cases of general paresis of the insane, in the severer cases of chorea, or in certain cases of intracranial syphilis, as may be seen by turning to the chapters upon these subjects; indeed, it would seem that a part of this description—that relating to the hæmatin and the tortuosity and the aneurismal swellings of the arterioles—is acknowledged to be from the work of Laborde upon softening and congestion of the brain of the aged, written in 1866. In addition to this description, Dr. Hammond also says that we will find this state of the brain described by Calmeil in 1826, subsequently given the name of *l'état criblé* by Durand-Fardel in 1854, and described respectively in treatises upon the insane and the aged.

The symptoms of spinal congestion are said to be: a dull, aching pain, increased by the recumbent posture and by standing, if the lower cord is affected, but not augmented by pressure; an occasional sensation of heat in the cord; disturbances of sensibility and motility, such as hyperæsthesia, shooting-pains, girdle-sensation, formication, etc.; erections, vesical and rectal paralysis, diminution of electro-

muscular reaction, and bedsores. These are the symptoms of myelitis; and not only has it never been proven that mere congestion can produce these symptoms, but there has never been any means demonstrated of making the differential diagnosis. The symptoms of spinal anæmia are said to be manifold. In one class of cases the morbid action has been supposed to be largely confined to the posterior columns of the cord. In another the anæmia is supposed to be restricted to the antero-lateral columns of the cord. It is admitted by the writers upon this subject that post-mortems are lacking to support these claims, and if the chapter upon "Combined Sclerosis" is read, together with the chapters upon "Lateral and Amyotrophic Lateral Sclerosis," it will be seen that the symptoms of disease of the postero-lateral columns of the cord are perfectly well known at the present day, so that if any congestion of them had been capable of causing the alleged characteristic symptoms just described, it should have been demonstrated by at least one post-mortem.

The truth of the matter is that our knowledge of nervous diseases has increased so rapidly within the last quarter of a century as to make it a matter of little surprise that the older authors should have attempted to satisfy their ignorant consciences by dubbing as congestion or hyperæmia the many puzzling symptoms which time has resolved into distinct system-groups. The most difficult of all phrases for the average scientist is, "I do not know." The disease must be labelled at any cost, and, once labelled, it takes many years to rub out the brand. Meningitis, general paresis, porencephalus, cerebral and spinal syphilis, disseminated sclerosis, bulbar lesions, poliomyelitis anterior, central or transverse myelitis, syringomyelitis, progressive muscular atrophy, acute ascending or Landry's paralysis, locomotor ataxia, especially in its early stages, many forms of neuritis, focal diseases of the brain, lateral sclerosis, many forms of lithæmia and neurasthenia—these are some of the many diseases of which we now possess a knowledge at which the general practitioner of twenty-five years ago might well stand appalled, and which he may well be pardoned for having supposed to be due to some vague form of anæmia or hyperæmia. Indeed, Leyden, writing in 1875 of essential infantile paralysis, thought it was caused by a peculiar congestion of the cord, while Cornil and Lépine had demonstrated some two years before that the pathological condition was a disappearance of the ganglion-cells in the anterior horns of the spinal cord, the autopsy in their case being followed by another one to the same effect by Gombault in the same year as Leyden's article.

After this necessarily cursory review, we may ask ourselves, Can hyperæmia or anæmia of the brain and cord be diagnosed? I, for my part, would answer very positively that a diagnosis is not possible by means of the nervous symptoms alone. In other words, the symptoms as I have narrated them above are not sufficient, individually or in a group, to warrant a diagnosis of anæmia or hyperæmia of the brain or cord. It must be remembered that the brain is an exquisitely and well-nigh inconceivably complex organ, pulsating and vibrating with a most explosively nitrogenized molecular life.

and reacting to myriad influences in a kaleidoscopic way that constitutes the never-tiring charms of character, poetry, music, and the varied forms of mental activity. To put our finger on a few slight changes of temporary nature that occur in an organ like this, say that they are really due to hyperæmia or anæmia, and exclude all the cellular play, all the reflex modifications from the other viscera of the body, all the morbific elements that may be circulating in the blood, all the factors of temporary discomfort that are to be found in the atmosphere, in the food and clothing, and in the circumstances of a human being's environment—even if this is not impossible, it has certainly not been done. If we have evidence of some intracranial disturbance, such as those we have already detailed—headache, delirium, vertigo, tinnitus aurium, insomnia, flushing or pallor of the face—and with these we have some concomitant conditions that would make it reasonable to suppose that there might exist a congestion or anæmia of the brain or cord, then we should be warranted in regarding such a diagnosis as probable. Thus, certain cardiac conditions, lesions of the great vessels of the chest and abdomen, certain forms of hepatic disease, tumors making pressure upon these great vessels, a general anæmia, leucocythæmia, intense mental exertion, or exposure to great heat—these conditions when associated with the above-described general symptoms, would make it probable that the general symptoms were due to hyperæmia or anæmia as the case might be; but even then there might not be certainty. I cannot conceive, however, how we are to make a diagnosis of spinal anæmia or hyperæmia, for I know of no pathological observations that would warrant on the one hand the diagnosis by means of the vague pains that are described by some authors, or, on the other, by the symptoms of the full-fledged diagnosis which others would have us place faith in.

THROMBOSIS OF THE INTRACRANIAL SINUSES.

There are certain peculiarities in the anatomy of the cerebral sinuses predisposing them to thrombosis, and these are their great width, the rigidity of their walls, their triangular form, their occasional septa, the fact that they are not emptied during inspiration, and, in the case of the longitudinal sinus, the entrance of the blood from the cerebral veins at an obtuse or right angle, so that it is opposed to the current. These factors all favor coagulation. There are two varieties of thrombosis in these veins, namely, primary or marasmic, and secondary, infective or inflammatory.

The marasmic form is much the least frequent, and is generally found in the longitudinal sinus, and rarely in either the lateral or the cavernous. It occurs chiefly in those who have been wasted by exhausting diseases, and is most frequent at the two extremes of life, in the very young and the very old. Exhausting diarrhœa is the most frequent etiological factor in children, but pulmonary affections, both acute and chronic, not infrequently cause it. Other supposed causes in children are respiratory obstruction with consequent dilata-

tion of the right heart, as from pneumonia, pleurisy, and tuberculosis. It is most frequent toward the end of these diseases or in early convalescence. In adults, it may be caused by enteric fever, phthisis, the puerperal condition, and occasionally by carcinoma in old people.

Marasmic thrombosis is generally confined to the longitudinal sinus, but it may extend to the lateral, sigmoid, and cavernous ones and the jugular vein. The clots are dense, resilient, stratified, and do not adhere to the walls of the vein, and seldom occupy the whole lumen of the vessel. Their organization is strong, and they rarely disintegrate, although in some chronic cases they may have a canal sufficient to permit the re-establishment of the circulation. Great congestion of the meningeal and cerebral vessels speedily ensues, with œdema, but, of course, these phenomena depend upon the site and extent of the clot. For instance, if this is confined to one sinus, part of the blood may still pass by some of the tributary veins. If the clot, however, is one of the cerebral veins, great œdema and congestion results, and the capillaries may become turgid and burst, giving rise, with the other factors, to red softening. The ventricles may even become distended with serum, and sometimes the retro-ocular tissue becomes the site of transudation, causing exophthalmus. The parietal and occipital lobes are most affected by secondary softening, whilst the frontal lobes are least implicated.

The symptoms of the disease are often obscure, and when present they depend, of course, upon the sinus that is affected. When the longitudinal sinus is implicated there is distention of the frontal and parietal veins, with œdema, occasional epistaxis, frequently strabismus, tremor of the lower limbs, contracture, and rigidity of the muscles of the extremities and sometimes also of those of the neck and back. General convulsions, with unconsciousness, are apt to occur in children, whilst in adults their place is taken by headache and delirium. It is said that the convulsions are occasionally unilateral or even monoplegic. They are apt to be intermittent in character and occur in paroxysms.

The diagnosis of marasmic thrombosis is not always an easy matter. Headaches, the paralysis of one side of the body or of a limb, with delirium, somnolence, coma, and œdema and venous distention of the scalp of the parietal and frontal regions, may create a strong suspicion of the affection in adults if other cerebral lesions can be excluded. (See chapters on "Cerebral Tumor," "Intracranial Tumor," "Brain Syphilis," "Hemorrhage," "Embolism and Thrombosis," and "Meningitis"). In children the tentative diagnosis may be made when an exhausting illness is succeeded by convulsions, œdema, and venous congestion of the scalp in the parietal and frontal regions, epistaxis, and evidences of some localized brain lesion.

Secondary infective or inflammatory thrombosis is caused by pathogenic micro-organisms, and occurs chiefly in adults, and seldom in old people or children. It generally affects one of the dual sinuses, and usually that one which is nearest to the seat of the primary disease. It is often associated in this latter stage with meningitis, as well as with small cerebral or cerebellar abscesses. The infection

may extend from some traumatic lesion of the scalp or cranium; but the most frequent cause is middle-ear disease affecting the sigmoid sinus. Pathological lesions of the face and head may also give rise to it, such as anthrax, erysipelas, cellulitis of the orbital cavity, ulceration in the nasal fossa and pituitary mucous membranes, periostitis of the jaw from dental caries, inflammation of the tonsils, and retro-pharyngeal abscess. The infection may extend to the sinus by contiguity of tissue, so that the wall of the vein becomes inflamed, and this in its turn sets up coagulation and adhesion of the clot to the inner coat; or a small vein at the site of lesion may become thrombotic, and thus the coagulation with its attendant phenomena extend to the sinus. In some cases pressure on the sinus, as from a tumor, is said to be the cause, but it is rare. In infective thrombosis the endothelium of the vein desquamates, and the walls become soft, thick, and so altered as to tear. Hemorrhage, singular to say, is nevertheless rare, because the blood coagulates simultaneously with the alterations in the coats of the vein. The clot of infective thrombosis soon disintegrates and breaks down into a greenish-brown grumous fluid swarming with bacilli. From this source infection may extend in innumerable directions, either through the veins or through the general circulation or through the coats of the vein into the neighboring tissues. The bone near the sinus may become deeply pigmented, of a dark greenish and brownish hue, and eroded, the staining being thought to be due to the action of the bacillus pyocyaneus or some other chromogenic organism, and these markings on the bone often photograph the position of the sinus very accurately. In some cases a similar stain may be left upon the cerebellum by the lateral and sigmoid sinus, or upon the bones and soft tissues by the internal jugular, posterior condylar, and vertebral veins. If the infective process has caused only a very limited disintegration, a formative process can be established in the wall of the vessel, granulations springing from the wall of the sinus and the adjacent dura mater, and penetrating into the osseous erosion on the one side and into the lumen of the vessel on the other. With time, fibrous tissue will completely obliterate the sinus. If the granulation-tissue should be displaced or disintegrated, the infection may pass through the open mouths of the vessels into the general vascular system, so that cerebral and cerebellar meningitis of the pia may occur, and even pulmonary infarctions, cerebral and cerebellar abscesses may be formed. The symptoms of infective thrombosis are themselves rarely sufficient to base a diagnosis on, but this can usually be done when there has been a history of trauma, middle-ear trouble, or some well-marked infective cause such as anthrax or facial erysipelas. Severe headache is an early and frequent symptom. It may be general, or over the region of the affected sinus, although its value as a localizing symptom is not always great. Vomiting is frequent. The febrile rise is rapid, occasionally to 103° or even 106° F.; but it has marked fluctuations and is sometimes very remittent. The pulse is small, quick, and thready, even when the patient is deeply narcotized, as by chloroform or ether. Rigors are frequent, coming early,

often repeated, and in some cases with a tendency to periodicity. They are succeeded by profuse perspiration, the tongue is dry and coated, and there is a heavy, peculiar odor from the mouth. There is a strong tendency to diarrhœa, although exceptionally there may be constipation. If the disease progresses, the symptoms may assume the pulmonary, abdominal, or meningeal type. The pulmonary symptoms are, first, dyspnœa, a localized stitch, "prune-juice" expectoration, and coarse râles, which are succeeded by moist râles. In the abdominal type there are abdominal pain, meteorism, diarrhœa, "pea-soup" stools of an extremely offensive odor, great prostration, pale, pinched face, often a bright blush of the cheek, occasionally a rather dark rash, like measles, which does not disappear on pressure and is not raised above the level of the skin; but there are no rose-colored spots, and, unlike the disturbance of the skin in enteric fever, the rash is not elevated, does not disappear on pressure, and does not come out in successive groups. The meningeal symptoms are the common ones of meningitis (see Chapter on "Cerebro-spinal Meningitis"), namely, vomiting, fever, great excitement, hypersensitiveness, and agitation, clonic and tonic contractions or paresis of certain groups of muscles, especially of the face, neck, and upper extremities, strabismus, retraction of the head backward or to one side, and delirium. The mind is clear, however, at the beginning of the trouble, although there may be some slight irritability. These three great types, pulmonary, abdominal, and meningeal, may be commingled. To them also may be added the symptoms of cerebral abscess.

TREATMENT. The treatment should consist in the removal of the cause, if possible, the remedying of the meningitis which may have ensued, and of opening the resultant cerebral or cerebellar abscess if there has been any. The treatment of the meningitis has already been discussed in the chapter upon "Cerebro-spinal and Suppurative Meningitis" and that upon "Abscess." The opening of the sinus itself and of the mastoid cells is a matter of surgery, for which recourse must be had to the general or aural surgeon.

SUPPURATIVE MENINGITIS.

Cerebral meningitis occurs in the different forms of tubercular meningitis, pachymeningitis, lepto-meningitis, cerebro-spinal meningitis, and hydrocephalus. Suppurative meningitis is the only meningitis of which we have any practical knowledge besides these.

ETIOLOGY. The best known causes are purulent pleurisy, ear disease, nasal disease, cerebral traumata, exposure to cold, insolation, infection, erysipelas, carbuncle, and extension from other viscera.

Ear disease is probably the most frequent of all the causes of suppurative meningitis. Between the ear and the petrous portion of the temporal bone there are many intercommunications by means of veins, arteries, and connective tissue. The latter is a particularly good avenue for septic extension, as it contains large branches of the middle meningeal vein and artery, passing to the mucous membrane

of the tympanum through the petro-squamous fissure. Another excellent highway for suppurative affections between the ear and the base of the cerebrum is the facial nerve, as only a very thin layer of bone separates it from the tympanic cavity, so that both the peripheral and central tract of the nerve may very readily be affected.

The cribriform plate of the ethmoid may conduct a lesion of the nasal bones to the brain, and in this way cases of meningitis following pertussis may arise.

Suppurative meningitis may be set up by cerebral traumata, and in so many instances there may be elicited a history of cerebral trauma which the family had entirely overlooked that I am inclined to regard this as a much more frequent cause than is usually supposed.

In rare cases exposure to cold and insolation have been known to act as causative agents in suppurative meningitis.

In some cases erysipelas and carbuncle are known to have been followed by suppurative meningitis.

Cases of suppurative meningitis have been observed following ulcerative endocarditis, septicæmia, pyæmia, croupous pneumonia, suppurative pleurisy, acute articular rheumatism, typhus, dysentery, scarlatina, and smallpox, as well as from suppurative disease of other viscera by the passage of the infection through the lymphatics or bloodvessels.

Pathological Anatomy. In meningitis resulting from nasal and aural disease the base and lateral aspects of the brain are mainly affected, whilst in the infectious cases the vertex is more implicated. In the slighter degrees congestion of the pia may be observed, dotted with purulent matter; but there is more or less thorough infiltration matting down the pia to the cortex, cloudy-yellow or greenish-yellow in color. Underneath this infiltration will be found flattened and ischæmic convolutions. Along the course of the bloodvessels this infiltration is most noticeable. It generally passes into the ventricles along the choroid plexus, and an increased amount of cerebro-spinal fluid is observed. Atrophy of the cortex usually results in chronic cases by reason of the membranes becoming tough and thick and adherent to each other. Micro-organisms are revealed by the microscope in these cases, and further examinations can be had by inoculations with or without culture. Netter has recently made a collection of forty-five cases of other authors and twenty-five of his own. In the latter were found six species of micro-organisms—in four, a streptococcus pyogenes; in eighteen, pneumococci; in two, a microbe resembling the intracellular diplococcus of Weichselbaum; in one, a microbe very much like the pneumo-bacillus of Friedländer; in one, a short bacillus of great mobility presenting most of the characteristics of the typhoid bacillus; and in one, certain bacilli that were very delicate and flexible. Netter's observations and those of the authors in the forty-five cases collected by himself tallied in the main. Netter believes that the character of the exudation is indicated by the micro-organism causing it. For instance, the exudation in which the pneumococci are found is greenish and viscous, and, although the affection is comparatively

a benign one, the meningitis almost always coincides with ulcerative endocarditis. The exudation was less adherent and of a sero-purulent nature in the cases in which the streptococcus was present, but remarkably thick and viscous in those cases containing the bacillus of Friedländer.

SYMPTOMATOLOGY. Suppurative meningitis is indicated by coma, fever, delirium, muscular twitchings, headache, convulsions, paralysis, and optic neuritis.

Many forms of cerebral affection are characterized by the symptoms of delirium, hebetude or coma, convulsions, and muscular twitchings, so that they are not pathognomonic of this particular form of meningitis. The degree of fever is usually between 39.5° and 40° C. A constant headache, intermitting to some extent, is always present. Paralysis may vary in its nature according to the portion of the brain affected, although it is usually present in the form of hemiplegia. Optic neuritis is sometimes seen, when it is of considerable diagnostic value; but it is often absent, and in most cases is not generally observed until the case is quite advanced. It does not indicate the character of the lesion, however, but simply indicates the fact that an intracranial lesion exists. Opisthotonus may even be observed in some cases, and retraction of the head usually occurs. In some cases the headache, dizziness, and some mental confusion, resulting in children from suppurative ear-trouble, may last for several days, and be entirely relieved by the discharge of pus from the external ear. In cases of less degrees of suppurative meningitis, particularly those caused by ear-trouble, the symptoms may commence with heaviness, sleeplessness, and slight headache, passing gradually into the more pronounced stage.

DIAGNOSIS. Cerebral symptoms alone cannot be depended upon for diagnostic purposes, but the presence of those causes most likely to occasion this type of meningitis must also be taken into consideration. When insolation or trauma is known to precede symptoms of meningitis in a child, it may reasonably be supposed that the meningitis is of a suppurative character. In any doubtful case a careful examination should always be made of the heart, the lungs, the nostrils and ears, and it should be ascertained whether the child has recently suffered from pertussis, typhus, croupous pneumonia, typhoid fever, cerebro-spinal meningitis, ulcerative endocarditis, purulent pleurisy, pyæmia, acute articular rheumatism, dysentery, scarlatina, or small-pox.

The diseases from which the diagnosis should be made are typhoid fever, tubercular meningitis, and cerebro-spinal meningitis.

The diagnosis from typhoid fever may sometimes be extremely difficult, unless sufficient time has elapsed to mark the variations of temperature, or unless the cutaneous or enteric symptoms manifest themselves.

The presence or absence of tubercular history in the family will aid in the differentiation from tubercular meningitis.

Cerebro-spinal meningitis, if sporadic or epidemic, may be differentiated by this fact from suppurative meningitis.

Prognosis. In cases which follow diseases of the middle ear or of the nasal cavities the prognosis is extremely uncertain, although many cases recover. When the disease occurs by metastasis from other viscera, there is not only great danger to life, but a still greater probability of cerebral or motor defects resulting. But the prognosis is usually fair in uncomplicated cases.

Treatment. The cause of suppurative meningitis will very greatly indicate the treatment. Operative procedure should be resorted to in cases of pent-up pus resulting from diseases of the ear or its appendages. If caries of the nasal bone exists, it should be promptly removed, provided it is of a nature to permit removal; but even in such a case it is of the utmost importance that the meningitis itself should be properly treated. Ergot is a remedy of great value, and should be given in the form of the fluid extract, every three or four hours in doses of from five minims to a drachm, according to the age. A few large doses of quinine, say from two to ten grains, according to the age, will often prove of service if the meningitis is subjected to treatment at an early stage. But its use after the early periods has not convinced me of any further efficacy, except as a tonic. Doses of from one-twentieth to one-tenth of a grain of the sulphide of calcium, every hour for a day or two, have seemed to me to be of considerable benefit. The restlessness incident to the disease may sometimes be relieved by cold applications to the head, or, if necessary, by occasional doses of the bromides. I have never satisfied myself that inunctions of mercury—usually of the unguentum hydrargyri—are of any especial use, time-honored though they are.

CEREBRO-SPINAL MENINGITIS.

History. Vieussens wrote the first description of cerebro-spinal meningitis in 1805. Whether it had occurred previous to this time is unknown, but shortly after Vieussens' account epidemics of it were observed in Europe. The first American cases were seen in Massachusetts in 1806. Since then there have been numerous epidemics. Thus, between 1816 and 1828 there was an epidemic in Connecticut, at Middletown, and another at Vesoule, in France, both being limited and brief. In 1828 it occurred in Ohio, in Trumbull County; in 1830 at Sunderland, England, and in 1833 at Naples. In 1837 it spread from the south of France over most of the kingdom, and continued at intervals until 1849, and also extended to the various places around the Mediterranean, and to Denmark, England, and Ireland. In 1842 it appeared in the United States in various places; again in 1843, and from 1848 to 1850. Between 1850 and 1854 there was no epidemic in any part of the world, but from 1854 to 1860 there was a severe epidemic in Scandinavia. Since 1860 it has appeared in nearly every civilized country, and it is the opinion of competent observers that it has become naturalized in the cities of the United States, varying in severity from time to time. In 1872 an unusually severe epidemic attacked the city of New York, extending even to the lower animals.

ETIOLOGY. Among the causative factors of cerebro-spinal meningitis are:

Season;
Anti-hygienic conditions;
Contagion;
Depression of general health;
Sex;
Age.

The winter season is that in which most cases are observed. Thus Lewis Smith states that of 166 epidemics, all but 50 occurred in the six months commencing with December; while Hirsch affirms that 57 epidemics in Central Europe occurred in winter or in the winter and spring, 11 in the spring, 5 between spring and autumn, 4 began in the autumn and continued into the winter or into the winter and the ensuing spring, and 6 lasted the entire year. But Smith thinks that in New York City season appears to exert little or no influence on the prevalence of the disease—because, as he believes, of the fact that New York tenements are under better sanitary regulation than the dwellings of the European peasantry.

It is certain that anti-hygienic conditions are favorable to cerebro-spinal fever, because it has been observed most severely in large numbers of people crowded together, as soldiers and tenement-house dwellers. A chart prepared by Moreau Morris, of the New York Health Board, seems to show that almost all the cases occur in those portions of the city where the sanitary conditions are bad.

Although it has often been impossible to detect the exact relation of one epidemic to another, yet there is excellent proof to show that the disease is slightly contagious, and this was notably shown in the epidemic of 1872 in New York, where the lower animals were affected, and in 1811 in Vermont, when foxes died of it.

Depression of the general health from overwork, bad air, insufficient food, privations of soldiers, etc., would certainly seem to predispose to cerebro-spinal fever.

There does not seem to be any predisposition of one sex to the disease, except, of course, that men, who are more exposed than women of the corresponding family, are more liable to break down with it.

According to the elaborate statistics of Lewis Smith, three-fourths of the cases have been under the age of ten years, and the following are the figures of the New York Health Board of the age of the patients in the epidemic of 1872:

Under 1 year	125
From 1 to 5 years	336
" 5 " 10 "	204
" 10 " 15 "	106
" 15 " 20 "	54
" 20 " 30 "	79
Over 30 years	71
Total	975

CLINICAL HISTORY. The onset is usually sudden, although in certain rare cases there may be prodromal symptoms for a few

hours or days, such as chilliness, lassitude, anorexia, etc. The onset is usually with headache, emesis, and slight chill or clonic convulsions. Coma and delirium usually succeed, or, in the slight cases, simple drowsiness. The temperature ranges from $99\frac{1}{2}°$ to $101°$, and as the disease progresses it may rise to $102°$, and even higher, some fatal cases going to $107\frac{2}{5}°$. The pulse is usually quickened, although it may sometimes be subnormal. When it is increased, it rises to 110, 120, and even higher. The face is generally pale, later on it is flushed, and often the *tache cérébrale* of Trousseau is observed; *i. e.*, a congestion of the skin produced by running the finger across the forehead, cheek, or ear, lasting for a short time. The skin may also present the characteristic appearance called "goose-skin," due to papilliform elevations, from contraction of the muscles of the corium; or it may be duskily mottled, or there may be numerous minute extravasations, which are occasionally so large as to be an inch or more in size. In some epidemics these appearances are not observed. Then there is quite commonly herpes, either early or after a week or two, and erythema and roseola occasionally occur. The skin may either be dry or perspiring. The urine is usually normal, or there may be polyuria, and the urine may contain a small amount of albumin, or there may even be cylindrical casts and blood-corpuscles. There is occasionally œdema around one or more joints. As the disease progresses more marked organic symptoms appear. Of these, the most common are strabismus and alterations in the pupil, such as contraction, inequality, feeble response to light, etc. Inflammatory changes sometimes occur in the media of the eye. The headache is usually severe, and attention is frequently called to it by the patient, who puts the hand to the spot with an exclamation of pain. Hyperæsthesia of the surface of the skin is common. Retraction of the head is usually marked, and there is frequently also contraction of the posterior muscles of the trunk, abdomen, and lower extremities, so that opisthotonus and flexion of the thighs and legs are common. These usually occur about the fourth or fifth day, although they may supervene as early as the second. Paralysis of the muscles of the extremities is also seen, and sometimes very shortly after the onset. This paralysis may be of one side (hemiplegic) or of both legs (paraplegic), or even of individual groups of muscles, although this last is rare. Choreic or choreiform movements do not often occur. The respiration is usually not greatly affected so far as regularity is concerned, although it may rise to forty respirations per minute. There is often mild otitis media, usually not leading to impairment of hearing, and this is generally bilateral. The duration is very variable, exceptional cases terminating in twenty-four hours, others in a week or two. When there is recovery, however, it generally takes place in three or four weeks, although fatal cases may last as long.

PATHOLOGY. Cerebro-spinal meningitis is a constitutional malady, of which the meningitis is simply one of the local manifestations. It is undoubtedly due to a micro-organism.

The meninges are markedly hyperæmic, although the extent of

this hyperæmia varies greatly in different cases—in some extending over most of the cerebral surface, in other being limited to small areas. The very acute fatal cases have not time to pass beyond this stage. Usually, however, free exudation is found in and beneath the pia mater, usually either serous, fibrinous, or purulent. Softening and cerebritis are usually observed.

PROGNOSIS. About 50 per cent. of the cases of cerebro-spinal meningitis are fatal, and of 113 cases which I have seen, in 30 there has followed some irreparable damage to the nervous parenchyma. Mental impairment is not nearly so apt to result as is paralysis, however. Those cases in which the clinical symptoms denote that the base of the brain is mainly affected are those in which an excellent prognosis can be drawn, so far as regards the mind; but even in these the speech-defects and paralysis of the members are often so great as to sentence the patient, especially if a child, to such a life of isolation and helplessness as to make the outlook very serious. Nevertheless, many cases do recover without either mental or physical impairment. The remissions of the disease are generally ominous. Unfavorable symptoms are—the occurrence beyond thirty, a very acute onset, coma, convulsions, active delirium, high temperature, numerous petechial eruptions, and continued albuminuria. Complications in other organs, either pulmonary or cardiac, are also of unfavorable prognosis.

DIAGNOSIS. The diagnosis of cerebro-spinal meningitis should be from simple meningitis and tubercular meningitis. In simple meningitis the onset is apt to be more acute, the hyperæsthesia and retraction of the head are less pronounced, and the presence or absence of a prevailing epidemic will throw great light upon the matter. In tubercular meningitis there is a prodromal stage of several days, and it may be preceded by symptoms referable to tuberculosis elsewhere.

TREATMENT. The patient should, of course, be kept perfectly quiet, the same attention being paid to keeping him sheltered from disturbing influences as has been recommended in the other forms of meningitis. Cold cloths should be placed on the head, taking care, however, not to have them icy, or not to refrigerate the patient with them. The drugs which are of most use are ergot, iodide, bromide, quinine, opium, and chloral hydrate. Ergot is, in my opinion, a very valuable drug in all forms of meningitis, and I believe that its value is heightened by the constant use of the iodide of potash and quinine. The fluid extract of ergot should be used, and it should be administered in half-drachm or drachm doses every three to five hours, according to the age of the patient. If the disagreeable taste of the fluid extract is objectionable, ergotine may be employed in doses of 2 to 3 grains, every three to five hours, as before, although I do not believe that it is nearly as efficacious as the fluid extract. The iodide of potash should be given in doses of 5 to 10 grains, three or four times a day. The form of quinine employed should be the tannate for young children and infants, and the sulphate for adults, the former in doses of 2 or 3 grains three or four times a day, and the

latter in doses of 2 or 3 grains three or four times a day. Some authors speak well of opium in this disease, but I have never been able to satisfy myself of its beneficial effects. If there are much restlessness or convulsions, or much delirium, the bromide of potash should be used, varying, according to the age of the patient, from 5 to 20 grains, three or four times in the twenty-four hours. In some cases, where the treatment by these four drugs does not seem to quiet the patient, moderate doses of opium or morphine will be found useful, preferably the sulphate of morphine, hypodermically, in doses of $\frac{1}{12}$ to $\frac{1}{8}$ grain once or twice a day, or even oftener. If in spite of these drugs there is insomnia, the best remedy, in my opinion, is, first, urethan, 5 to 10 grains at bedtime, in solution in water, or, if this does not answer, sulphonal, 5 to 15 grains at bedtime, in the form of a reliable compressed tablet, which should be tested by the physician to ascertain that it is perfectly friable. Besides all these, however, the most careful attention should be paid to the nursing and to the diet, which latter should be suitable to the age of the patient, and always be bland and easily digested.

TUBERCULAR MENINGITIS.

History. Robert Whytt, in 1768, first precisely described tubercular meningitis under the name of hydrops of the cerebral ventricles.

Clinical Symptoms. The prodromal symptoms are usually vague. The children are prone to emaciate, to be pallid, peevish, listless, and lose their interest in the thousand and one things that interest children. Sleep may be interrupted, and the child may awake complaining of headache in some one spot. The tongue may be coated, the patient may occasionally vomit, and the vomiting may be propulsive or like a regurgitation. There may be some constipation, the urine may diminish, and there may be some slight hyperæsthesia of the abdomen. There may also be photophobia or a tendency to sleep. Usually these symptoms increase in intensity, and the temperature rises from $100\frac{1}{2}°$ to $101\frac{1}{2}°$. The pulse, which has been slightly irregular, becomes much more so. These prodromal symptoms may last one or several weeks. Then more pronounced symptoms appear. Respiration becomes interrupted, and the patient sighs deeply and long; the abdomen is retracted; the eyes half close, giving the peculiar aspect that is often seen in dying people, and the eyeballs may move slowly in a lateral direction. One or both pupils dilate. There are sudden flushes of the face in limited areas, and the skin generally presents a red mark when the finger is pressed over it or when it is pressed upon the pillow, and friction causes a blush corresponding to the area rubbed (the so-called Trousseau's mark). Then there may suddenly break upon the scene more marked evidences of organic implication, such as slight twitchings around the mouth and eyelids, or a convulsion, usually generalized, followed by ptosis, amaurosis, strabismus, or paralysis of the face or extremities, usually unilateral. In some rare cases the paralysis may affect one extremity alone. The temperature now rises to 102°, 103°, or more,

the head becomes retracted, there is general perspiration, the pulse increases in frequency, and so the symptoms may increase until death, after a period of three or four weeks. Death is usually preceded by Cheyne-Stokes respiration.

In some children the symptoms may be much milder, and convulsions may not be observed until a few days before death. In this form vomiting, headache, delirium, convulsion, are very marked, and it is said that a tumid abdomen may be present instead of a retracted one, and the onset may be very sudden. In other children there may be so much somnolence from the start as to render the diagnosis very difficult. Occasionally there is a hyperæmia of the retina or tubercles of the choroid.

Etiology. The etiological factors of tubercular meningitis are heredity, age, and erythema nodosum.

A child coming of tuberculous parents is much more likely to have tubercular meningitis than one of healthy parentage, and yet tubercular meningitis cannot be said to be hereditary.

Most cases are observed between the second and seventh year.

A number of cases connected with erythyma nodosum have been reported.

Pathology. In tubercular meningitis the base of the brain is especially prone to be affected, and that portion of the base in which lie the olfactory, optic, and third nerves, and the crura cerebri. (See Fig. 36.) Changes may be found wherever the pia mater extends over the surface of the brain and into the ventricles The pia at the base, extending over the quadrangle that has just been described, is succulent, and looks like wet blotting-paper, very much as it does in syphilitic meningitis. Over the rest of the base of the brain and over the vertex where the pia is more closely approximated to the cerebrum, this membrane is opaque and thickened, but it cannot usually be detached easily, and it has the same characteristics in the choroid plexuses of the ventricles. There is usually exudation of a serous or purulent or gelatinous character. The ventricles will usually be found to contain an abnormal amount of fluid, which may be clear or turbid ; and when this fluid is excessive the ventricles are enlarged, and the basal ganglia, the fornix, and the corpus callosum may be softened. Tubercles are usually observed, either strewn around the vessels, or beaded through the pia like little grains of rice, or isolated so as to form genuine tumors, which are usually, however, of small size. The cranial nerves are implicated to a varying degree in the tuberculous and meningitic infiltration. A section of the pia mater and the subjacent cerebral tissue, made after hardening in alcohol and colored by Ehrlich's method, will show marked changes. The pia mater will be thickened, often extending like a wedge into the cerebral substance. There is often thrombosis of the arterioles and of the venules of the pia, obliterating the lumen. The walls of these vessels are thickened, often hyaline, surrounded by connective tissue and infiltrated with small round cells with distinct nuclei. Further away from the vessels areas of reticulated tissue will frequently be observed. Many bacilli and granulations will be found. Giant-cells

are sometimes, though rarely, seen in the internal wall of the vessel and in the surrounding cerebral tissue, and the tuberculous bacilli are strewn throughout the vascular walls. The perivascular sheath is often dilated and filled with lymphatic cells.

The discovery of the bacterium of tuberculosis was made by Robert Koch. He first found in the sputa of tuberculosis, in sections of miliary tubercles, and in tuberculous lesions in many organs, certain fine and elongated bacilli which colored in a peculiar manner. He discovered that they were especially frequent in the giant cells. Koch's observations have been confirmed by Balmer, Frenzel, Lichtheim, Gibbes, Charnley, Smith, Ransom, Heron, Prudden, Shakespeare, Ernst, and many others. Although Koch's observations have been attacked by certain experimenters, notably by Spina, of Vienna, yet they have met with general acceptation. In tuberculous meningitis, however, other bacteria have been found than those of Koch. In twelve cases examined by Babes, in seven there were found different microbes, and this variety explained, in his opinion, the acuteness and the purulent nature of some cases. In all these cases there were also tuberculous lesions of the mediastinal ganglia, and often pulmonary lesions of the same nature. In two cases there was a lanceolated microbe; in a third there was the ordinary streptococcus of pus; in the fourth there was the staphylococcus aureus; in the fifth there was also the streptococcus of pus, and in the two remaining cases there were peculiar pathogenic bacilli. In one of these cases there was a fine bacillus very closely resembling that of tuberculosis, and inoculation of it by trephining into the meninges of the rabbit produced acute meningitis and death in two days. In another the microbe resembled that of Friedländer, and inoculation of it also produced a meningitis in the rabbit. In another case there was a tetrahedric microbe in the pus, or arranged in groups like the staphylococci. Inoculation of this was negative. In still another case there was a peculiar microbe which developed an unusual colony in agar-agar. In other cases, however, that were examined by Babes, no microbe was found except that described by Koch. In two cases of meningitis caused by purulent otitis Babes has also found a few tubercular bacilli.

Prognosis. The prognosis of tubercular meningitis is very unfavorable, and cases rarely recover, if ever. I myself have never seen a case of recovery, but a few have been reported by different writers, such as Hahn and Abraham Jacobi, the latter of whom claims that he has seen two cases.

Diagnosis. The diagnosis of tubercular meningitis may be readily made when the symptoms of a tubercular meningitis supervene upon tubercular lesions that have previously existed in other organs. When however, there is no history of tuberculosis personally in the individual, and when the hereditary history is not pronounced, the diagnosis between tubercular and simple meningitis may become very difficult. The prodromal symptoms of gradual change, to which are superadded symptoms of organic affection of the cerebrum in the way of convulsions, paralysis of cranial nerves or extremities, the tem-

perature rising to 101° and above, the retraction of the neck, the half-open eyes, the delirium, the somnolency—all these make a picture of an intracranial affection that can be diagnosed as tubercular meningitis when other lesions capable of causing similar symptoms are excluded. These other lesions are:

Simple meningitis;
Syphilitic meningitis;
Cerebro-spinal meningitis;
Tumor;
Typhoid;
Trauma;
Ear disease;
Uræmia;
The hydrocephaloid state;
Mental disturbances from pulmonary lesions.

In simple meningitis the onset is more sudden, and the prodromal symptoms will be wanting. The temperature is higher, the disease runs its course more quickly, and there will be no history of tuberculous infection or predisposition.

Syphilitic meningitis occurring in an adult will be accompanied by symptoms of intracranial syphilis, and in the child with syphilitic heredity by such well known signs as the characteristic rash, chronic snuffling, usually beginning soon after birth, collapse of the bridge of the nose, early enlargement of the spleen, and rapid improvement with mercury.

Cerebro-spinal meningitis occurring as an epidemic is, of course, easily diagnosed. When it occurs endemically or solitarily, the prodromal symptoms are short, the characteristic eruption may be present, and the vasomotor disturbances evidenced by pressure upon the skin occur earlier.

Intracranial tumor in an adult or a child will be much more prolonged, have more localized symptoms, and will not have any temperature-rise.

Typhoid fever, when typical, may be distinguished by the temperature-curve, but when non-typical the diagnosis in the first week or two will frequently be absolutely impossible.

Trauma may set up a meningitis or a hemorrhage succeeded by a meningitis, and from this the diagnosis can be made easily enough if the trauma is known; or if not, the onset of the symptoms will be quicker and the general history will be more that of a simple meningitis.

In every case of meningitis in which the cause is not clear, the ear should be carefully examined, and any purulent trouble found there may aid in making the diagnosis plain.

Uræmia can be diagnosed by careful examination of the urine, and in the later stages by a lack of the organic symptoms present in a meningitis.

The hydrocephaloid state may present so many of the symptoms of a meningitis that for a time the diagnosis may be in doubt, but the history of some acute disease, especially some gastro-enteric

trouble, having preceded the brain-symptoms, will soon make the nature of the case evident.

It should always be borne in mind that an acute pulmonary lesion, especially in a child, may give rise to mental symptoms that may simulate a meningitis, but auscultation and percussion of the lungs will clear up the diagnosis.

TREATMENT. Although there is a doubt that has already been stated whether treatment is of much avail in tubercular meningitis, yet the very existence of a doubt renders it our duty to treat carefully and intelligently. The patient should be put absolutely at rest and screened from any disturbance that may impinge upon any of the peripheral nerves or the cerebrum. The room should be darkened, a careful and tactful attendant should take charge of the case, and medicines and food should be administered with clock-work regularity, for in dealing with a fatal disease every attention should be paid to the most minute details. The medicines administered should be the bromides, the iodide of potash, quinine, and opium. The best bromides are either the bromide of potash or the bromide of soda, and there is usually no choice between them, although in some exceptional cases the bromide of soda will agree best with the stomach. The bromide of potash should be administered in doses that should vary with the age and the amount of convulsive disturbance. In children 5 to 10 grains three or four times a day, are usually sufficient. In adults 15 to 20 grains three or four times a day should be used. In case of convulsions these doses should be increased according to the judgment of the physician. The iodide of potash should be administered in doses varying from 5 grains three times a day in children, and 10 grains three times a day in adults, to double or treble these amounts; and it should be given in a wineglass, or, better still, a tumbler, of water, or perhaps even in a glass of Vichy in cases where the stomach is irritable. Quinine should be given in the form of the tannate three or four times a day in children, and in the form of the sulphate three or four times a day in adults. The tannate can be best administered in the form of chocolates, which are made up nowadays by most of the manufacturing chemists; and the sulphate may be given in the form of a capsule or the tablet triturates, and never in solution with an acid. I am aware that there is considerable skepticism as to the value of quinine in any form of meningitis, and yet I am firmly of the impression that the strength of the patient holds out better when quinine is administered. Opium is a very valuable agent in calming the excitability of the cerebrum. In children it may be used in the form of 5 drops of laudanum once, twice, or thrice daily, as the physician may judge best; in adults, hypodermics of the sulphate of morphine, $\frac{1}{8}$ grain, of the aqueous extract of opium $\frac{1}{8}$ grain, may be used either by the mouth or hypodermically once, twice, or thrice daily, as may be indicated. In some cases moderately cold applications seem to give the patient great comfort, whilst in others they irritate, and they should therefore be used or not, as may be deemed best; but I disapprove entirely of the practice of putting ice-bags

or ice-cold compresses upon the patient's head, for fairly cold applications will answer every purpose of contracting the arterioles, whilst ice applications may dangerously lower the bodily temperature, and thereby seriously decrease the general strength. If there be persistent vomiting, the subnitrate of bismuth, 5 to 10 grains, with $\frac{1}{6}$ or $\frac{1}{3}$ grain of codeia, will be found of the most use; or a mustard-plaster applied over the epigastrium from time to time, or pills of ice or opiates. In case of cardiac weakness this should be met by a cardiac stimulant, such as digitalis, camphor, and strophanthus. Great attention should be paid to the food, and this should be of the blandest and most nourishing character and suited to the age of the patient. If there is persistent vomiting to such an extent as to interfere with alimentation, injections should be used.

CEREBRAL ABSCESS.

Definition. Cerebral abscess results from suppurative encephalitis.

Etiology. Abscesses are caused by traumata, caries of the cranial bones, nasal disease, intracranial tumors, and occur at times without any assignable cause.

Although marked symptoms may not supervene immediately upon a trauma, abscess may follow after a lapse of quite a period of seeming immunity.

Abscess may result from caries of the cranial bones, notably that of the petrous portion of the temporal bone, more particularly when it is conjoined with ear disease.

Nasal polypi and caries may cause abscess, the former sometimes causing disease of the frontal lobe by penetrating the cranial cavity.

When abscess is caused by intracranial tumors the latter are always in close proximity to the former.

Purulent lesions of other organs sometimes coexist with cerebral abscess, among which may be mentioned ulcerative endocarditis, pulmonary gangrene, bronchitis, empyema, puerperal fever, typhus, variola, scarlatina, and measles.

Symptomatology. Only vague symptoms manifest themselves at first. The child may show a decided malaise, complaining and being peevish, or marked headache may occur. Delirium usually succeeds these vague symptoms, or they may give place to hebetude and coma. The temperature does not usually range high, seldom going above 101°, and sometimes dropping to 99°. Convulsions occur in most cases. Vertigo and headache are usually present. All these symptoms culminate finally in paralysis, hebetude, and coma. To sum up, the symptoms progress steadily and slowly, and the individual manifestations will vary according to the location of the intracranial lesion.

Pathology. A localized encephalitis tending to the formation of pus is the direct cause of cerebral abscess. A limiting membrane may or may not surround it. Secondary changes may, of course,

take place in the contiguous cerebral tissue. Except in the pyæmic cases, abscess is generally single.

DIAGNOSIS. The gradual onset and variability of the symptoms, when conjoined with nasal or aural disease, should create a suspicion of cerebral abscess, or when such symptoms occur as a sequel to some one of the suppurative or infectious diseases of which a history is obtained, it should be differentiated from cerebral tumor or meningitis.

Tubercular meningitis is a chronic affection, either resulting from an existing tuberculosis or from hereditary predisposition. Cerebral abscess has a slow and insidious onset which distinguishes it from cerebro-spinal meningitis, and in the latter the retraction of head and abdomen, and the sporadic or epidemic presence of the disease, differentiate it from the former. As cerebral abscess and suppurative meningitis are often coexistent and proceeding from the same cause, it is not always possible to make the diagnosis.

Cerebral tumor is usually much more chronic than cerebral abscess, and optic neuritis is occasionally seen in it, never in cerebral abscess.

It will sometimes be possible to make use of the cerebral thermometer, to which I directed attention about twenty years ago. But it must be remembered that the slight changes of import in rectal and axillary thermometers are useless in cerebral thermometry, as here from three to five degrees are necessary to indicate anything abnormal. (See page 174.)

PROGNOSIS. Cerebral abscess is of very grave prognosis. The tendency to recovery idiopathically is so slight that an operation should never be delayed on account of this probability.

TREATMENT. When cerebral abscess is positively diagnosed an operation should at once be resorted to. Reference is made elsewhere in this work to anatomical data that may be used in localizing it. (*Vide* "Cranial Topography," page 97.) I have diagnosed two cases of cerebral abscess in the centrum ovale, in spite of the prevailing opinion that localizing symptoms alone can be depended upon for this purpose, and Von Bergmann has located one in the temporal lobe. The pain which follows percussion upon the skull may sometimes localize an abscess, but unless this symptom coincided with others of localizing value, I must confess that I should hesitate to follow this indication. I have, however, known these symptoms to conjoin in several instances. The localizing process will be materially assisted by the use of my cerebral thermometers. Every case of cerebral abscess should be operated upon, as great relief has often been the result of surgical interference. Von Bergmann reports a case where the patient had suffered from a purulent discharge from the right ear for fifteen years, caused by a cerebral abscess, which was cured by an operation.

SYPHILIS OF THE NERVOUS SYSTEM.

HISTORY. Syphilis seems to have been known first to the European world about the end of the fifteenth century, and the earliest

writer upon its connection with the nervous system is said to have been Leo Nicenus. Ulrich von Hutten first observed the relation between syphilis, apoplexy, and paralysis. Massar first called attention to syphilitic neuralgias, although Paracelsus had previously described syphilitic phthisis, diarrhœa, and hydrocephalus. Astruc, in 1740, spoke of syphilis as a probable cause of circulatory disturbances in the cranium. Van Swieten, Carrerè, and Swediaur attributed many chronic diseases to syphilis; but, singular to say, so keen-sighted an observer as Hunter positively stated that no brain or other viscus diseased by syphilis had ever been seen. This was in 1787. The enormous influence of Hunter's dictum paralyzed observations of this kind for some time, and the discovery by Morgagni, in 1779, of gummatous tumors of the cortex was entirely overlooked, and it was not until the time of the great Virchow that men uncovered their eyes and recognized the import of that which lay before them. In 1854 Gildemeester and Hoyack published a description of occlusion of the right Sylvian artery by a neighboring syphilitic formation. Then began a great strife between Robin and Wagner on the one hand, and Virchow on the other, as to whether these new formations were specific pathological products or whether they were new formations of granular tissue, Virchow maintaining the latter position. The solution of this question was due to the observations of a Dane, Steenberg, in 1860. Still the subject did not attract the attention that it deserved and that it has since obtained, although confirmations of Steenberg's statements were contributed by Griesinger, Passavant, Wilkes, Weber, and Wagner. The great work of Heubner, in 1874, however, conclusively confirmed Steenberg, and settled the matter beyond the shadow of a doubt, and especially did the former do this by his minute description of the peculiar syphilitic diseases of the intracranial arteries. Since that time the literature has become so enormous that, in a monograph written some five years ago, I was able to count up five hundred different articles on the subject in the preceding thirty years; and this, as I stated, was one-twentieth of the whole Sanskirt literature, or of the combined classical literature of Italy and Greece. It must suffice here to say that, salient among the salient, are Köster, Friedländer, Chvostek, Eichhorst, Greiff, Leyden, Yvaren, Gros and Lancereaux, Charcot, Hughlings-Jackson, Taylor, Moxon, Clifford Albutt, Fournier, Morel-Lavallée, Belières, and finally, in 1887, appeared the complete and decisive work of Rumpf.

Etiology. Syphilis of the nervous system usually occurs in the secondary and tertiary stage. Thus, of 150 cases of my own 87 have belonged to the tertiary stage, whilst 41 have occurred in the secondary and 22 in the primary stages. It will thus be seen that, contrary to the affirmations of those who have limited their observation of syphilis to the skin and mucous membranes, the nervous system may be affected at any time. Indeed, the great French syphilographer, Fournier, goes so far as to say that syphilis manifesting itself prominently in the cutaneous organs and mucous membranes is of the benign form, and is not so likely afterward to affect the nervous

system as is syphilis in which there have been scanty cutaneous manifestations; and this view is confirmed by my own observations as I have just stated them. Syphilis may be communicated in various ways. Thus, a chancre may be acquired in the ordinary fashion, or through the buccal membrane, through examinations by a physician, or through a surgical operation, and it is even a question as to whether it cannot be acquired through articles of clothing, whilst heredity is an occasional cause. Physicians should bear all these sources of infection well in mind, and should emancipate themselves from the traditional idea that it can only be acquired in the usual way. I have known several physicians who have infected themselves through examinations of patients or through surgical operations; and I also know of numerous cases in which the wife has been innocently infected through the husband. For all these reasons, it is evident that we cannot exclude syphilis because of a lack of a clear history of infection or because of a lack of the usual typical cutaneous manifestations.

In 150 cases of my own the average age was thirty-five, the maximum being fifty-two years and the minimum eighteen; 135 were males and 15 females.

PATHOLOGY. Syphilis of the nervous system may affect any portion of it—intracranial contents, spinal cord, peripheral nerves, and the membranes and bony structures surrounding the spinal cord and intracranial contents, as well as the arteries and capillaries within them. The arterial lesions were first described in 1874 by Heubner, of Leipsic, whose observations have given rise to further and even contradictory investigations by Köster, Friedländer, Baumgarten, Rumpf, and many others. Heubner advanced the view that in syphilitic vessels the endarteritis was peculiar in its place of origin, which was in the vessel-less tissue that lies between the fenestrated membrane and the endothelium, which two structures are normally so closely approximated that no intervening tissue can be seen microscopically. In syphilitic endarteritis, however, Heubner maintains, they are widely separated by a new cellular formation which consists of a proliferation of endothelial cells, to which, after a certain time, there are added numbers of peculiar round cells, of which we shall have more to say later. Heubner very minutely and carefully describes the differences in size of the endothelial cells in health and disease, as well as how to obtain an endothelium that has not been produced by the ordinary act of cutting the vessel, and it it difficult for anyone who reads his painstaking brochure to refuse credence to his plea for this peculiar anatomical site of syphilitic endarteritis; nor is it easy to resist the wish that it were true, as it would constitute so valuable a guide in dubious cases; but in the eighteen years that have elapsed since Heubner first made his views public no one has fully confirmed him. Köster, Friedländer, Baumgarten, Huber, Schottelius, Marchand, and Rumpf deny that the endarteritic process starts in the vessel-less structure between the fenestrated membrane and the endothelium, or that it consists at first of an endothelial proliferation, maintaining, on the contrary,

that this endarteritis is due to the outpouring of round cells from the minute nutritive vessels of the vessels themselves—from the *vasa vasorum*—contained in the outer coats, and that the endothelial proliferation is only a part of the general cellular disturbance that takes place in consequence of this outpouring of round cells.

The question needs further investigation. But, however it may be decided, there is no diversity of opinion as to the results which follow the endarteritis. The vessels may become narrowed in their lumen by the thickening inward of the fenestrated membrane and the intima. They may be entirely occluded from the same cause. The inner surface of the intima may become roughened or altered in its chemical constitution, and a thrombus may form, which, in its turn, may give rise to an embolus. It may even happen, though rarely, that minute aneurisms may be formed from the wasting of the muscular coat of the vessel, and occasionally there may be a hemorrhage. In other words, the syphilitic infiltration may set up an endarteritis, which usually leads to a narrowing of the canal of the vessels or to an occlusion of it, but it may occasionally induce thrombi, emboli, aneurism, and hemorrhages. Around the vessels will be found a number of cells reaching out into the tissue for a considerable distance, tapering off into the normal tissue and becoming scantier and scantier as the normal tissue predominates. The tissue in which these cells lie is finely granular, and is traversed by bands of connective tissue. This tissue is evidently the product of the cellular infiltration, with slight exudation. The cells are in all appearances similar to the white blood-cells, but their histological conduct is peculiar. They preserve their vitality for a great length of time in some places, and when they do undergo retrogressive metamorphosis it is into caseous and calcareous products and connective tissue, and seldom into pus—differing in these respects entirely from the cells that are found in tuberculosis, and acting rather like the cells of lupus, leprosy, and actinomycosis. Virchow classifies them with the granulation-tumors, Klebs with the infectious tumors, and Ziegler with the infectious granulation-tumors. Rumpf states that the cells that are near a vessel are very tardy in undergoing any retrogressive metamorphosis, and it is only those that are so far away from a vessel as to be deprived of their proper nutrition, either because of their distance or because of the disease in the vessel, that undergo degenerative changes. It matters not what portion of the nervous system is affected, the changes are always essentially similar in character, differing only in the form somewhat according to the histological peculiarities of the tissue affected. In the nervous system there is usually a general infiltration of all the different tissues, varying in degree in different parts.

To the naked eye these pathological conditions will present different pictures. The bones of the skull or vertebral column may be thickened, or there may be spicular or long pencil-like formations. The membranes are usually greatly affected, especially at the base. One of the most characteristic appearances is the infiltration of the pia mater at the base as it stretches along over the quadrangular space

that is formed by the pons behind, the tips of the temporo-sphenoidal lobes at the sides, and the posterior portions of the frontal lobes in front. (*Vide* Fig. 18.) Here the membrane will be found to be pultaceous, like thoroughly wetted blotting-paper, instead of presenting the translucent appearance that is usual of the normal pia mater. This is very much the same appearance as is presented in cases of tubercular meningitis; and, indeed, as we shall have occasion to see, there is no means of distinguishing between the two except by the accompanying history and the accompanying pathological findings. The membranes are usually adherent, so that the cerebral substance tears as they are pulled away. It is very seldom, however, that any purulent formation is observed. Studding the membranes in places may be found little granulations, and these are often large in size, constituting veritable tumors. The cranial nerves at the base, especially in the anterior portion that has been alluded to, are often found infiltrated by the diseased membranous structures, or surrounded by them, frequently both. In the spinal cord the periphery may be affected, or there may be infiltration of the whole cord, so that the process would seem to extend to the cord either through the membranes or the vessels. When the spinal or cranial nerves are affected there is an intense infiltration of the peculiar cells, with a degenerative process of many nerve-fibres, or there may be granulations or actual tumors of the nerves. In some cases the nerves attain to an enormous size, and the bony canals through which the nerves run may also be affected by osseous infiltration. Donné found a *vibrion vineola* in the secretion of chancre in 1837, without, however, attributing to it any importance. Hallier, in 1869, thought that he found multitudes of micrococci in the blood-globules. In 1872 Lostorfor saw on the third day, in syphilitic blood preserved in a humid chamber, small brilliant corpuscles with minute prolongation, these becoming larger and mulberry-shaped; but it has since been shown that these phenomena are observed in normal blood, especially in cachectic individuals. In 1878 Klebs found little rods animated by very slow movements in the liquid from a chancre, and, cultivating this liquid in gelatin, he developed these rods into agglomerations forming large spiral masses, which he called *helicomonades*. The injection of this liquid into apes produced circumscribed buccal alterations like mucous patches, and at the autopsy of one of these animals caseous deposits were found between the dura mater and the skull resembling gummata, whilst caseous patches were seen in the lungs, the pleura, the kidneys, etc. Other apes were injected by Klebs subcutaneously with fragments of chancre, and at the autopsy caseous deposits were found, and the cultivation of the blood of these animals again gave rise to the peculiar rods and the helicomonades. Cornil and Babes, however, doubt whether these caseous nodules might not have been due to tuberculosis, to which malady apes are peculiarly subject in northern climates. The next year Klebs confirmed these researches, but it is singular that he was never able to obtain these results from inoculation in any other animal than the ape. Aufrecht found microbes in the secretion of condylomata in

1881. Birch-Hirschfeld gave a description in 1881 of certain bacteria which he had found in chancre, condylomata, and visceral gummata. They consisted of rods varying in length from one millimetre to three or five, and were found in the cells or in the intracellular tissue. In 1882 Martineau and Hamonic stated that they had observed a multiplication of rods obtained by culture of fragments of chancre, and that inoculations of apes had produced eruptions comparable to those of syphilis, such as hard chancre and the secondary phenomena. Letnic (1883) obtained negative results in inoculation of the hog and the rabbit with the micrococci cultivated from chancre and mucous patches. Köbler, Neumann, Bayer, Horand, and Cornevin experimented upon a number of animals, and arrived at the conclusion that no animal had yet been found capable of receiving the syphilitic virus. Cognard (1884) inoculated an ape with the cultivated secretion of a mucous patch, and, like Martineau, obtained an induration at the point of inoculation and a general eruption, but the objection has been raised that this was probably a species of septicæmia. Morrison (1883) has found bacteria in the secretions of chancre and mucous patches. Tornery and Marcus (1884) have cultivated micrococci. Königer has seen bacilli in a case of pulmonary syphilis, these bacilli being somewhat finer and longer than those of tuberculosis, and not colorable by Ehrlich's method; but many objections have been raised to his observations both because of their lack of evidence in the results and because of the methods of cultivation and inoculation; and the same objections are pertinent to the observations of Schütz, who claims to have found similar bacilli. Lustgarten has observed, in indurated chancre and in gummata, bacilli that were isolated or in groups, resembling those of tuberculosis. They were sometimes somewhat curved and enclosed in tumefied lymphoid cells, resembling the bacilli of leprosy and tuberculosis. In a subsequent communication the same author stated that he had detected them in the lymphatic vessels and the capillaries. They are colorable by a method described by Lustgarten, who found them in sixteen cases of indurated chancre, mucous patches, and the secretions of tertiary syphilitic productions, as well as in the gummata of an infant afflicted with hereditary syphilis. Doutrelepont has seen these bacilli described by Lustgarten in eight cases of chancre of the prepuce, in one condyloma of the labia majora, and in five papules, and he has observed the greatest number of them in cases of syphilis that have not been treated. One indurated chancre without secretion did not contain them, but they were found in many of the sections. Alvarez and Tavel have found the Lustgarten bacilli in the smegma and the desquamation of the humid portion of the genital region, as well as upon the surface of syphilitic and non-syphilitic ulcerations of the genital organs, and from this it would seem at first sight that these bacilli may be found under normal conditions; but the method employed by Alvarez and Tavel is not identical with that of Lustgarten, as the bacillus described by the latter is not colored by simple colors or by Ehrlich's method, whilst the microörganisms of Alvarez and Tavel are; nor have the latter found their bacilli in sections.

Klemperer and Matterstock have confirmed the observations of Alvarez and Tavel. Cornil and Babes are undecided as to which of these authors is right. Other writers upon the subject on different sides of the question have been Giacomini, Disse and Taguchi (two Japanese medical men writing in German), Gottstein, Leloir, Weigert, Baumgarten, Eve, and Longuard. As a result of all these researches it may be stated that the bacilli described by Lustgarten and Doutrelepont have been the ones that have attracted most attention; that the question is not by any manner of means yet decided; that the bacilli described by Lustgarten have been found by many in syphilitic individuals; that the bacilli similar in some respects to those of Lustgarten are found in normal smegma and the secretions of the genital organs; that the bacilli of Lustgarten and those of the smegma and the genital organs differ from one another distinctly in several particulars; that there are probably several bacilli in syphilis, two varieties being described by Rumpf; that the bacilli of Lustgarten have been found in all stages of syphilis and in syphilitic alterations in many different diseases; that nothing like them has been found in non-syphilitic individuals except in the smegma or the genital secretions, and even here there are alleged points of difference. It would therefore seem as if Lustgarten's bacilli bore some relation to syphilis. No one has as yet succeeded, however, in obtaining any pure cultures of this bacillus, and this test is therefore lacking.

Clinical History. The symptoms of syphilis of the nervous system are almost coextensive with the symptoms of all nervous diseases, and it is, therefore, very difficult to give a clean-cut picture. In most cases, however, the symptoms are either of lesions of the intracranial organs or of the spinal cord. Lesions of the peripheral nerves are rare. We may, therefore, classify the symptoms of syphilis of the nervous system into those of—

The intracranial contents;
The spinal cord;
The peripheral nerves.

Intracranial Syphilis. In 1887, and again last year, I called attention to the fact that most forms of intracranial syphilis are characterized by the following group of symptoms, namely: a cephalalgia that is apt to be peculiar in having a quasi-periodicity that manifests itself in a tendency to return at a certain time in the twenty-four hours, most frequently at or toward night, less frequently in the afternoon or morning; marked insomnia, usually at the onset, lasting a few weeks; a sudden cessation of the cephalalgia and insomnia upon the supervention of any paralytic or convulsive symptoms. I have been working up this subject for some thirteen years, and I have a history of 140 cases. These symptoms are usually prodromal to the various lesions of the cerebrum that may be manifested. In some cases, however, these prodromal symptoms will be absent, although it is only in a minority that this will happen. Whether preceded or not by these prodromal symptoms, the lesions of the cerebrum will manifest themselves by symptoms of paralysis, coma, epilepsy, or insanity. The most usual forms of paralysis are either

hemiplegia, monoplegia, or paralysis of one or more cranial nerves. The hemiplegia may be produced by a lesion of the cortex, of the basal ganglia, or of the internal capsule, in none of which will there be any implication of the cranial nerves. Hemiplegia produced by cortical lesion usually begins by paralysis of the upper or lower extremity, which gradually emerges into a hemiplegia. Hemiplegia of the internal capsule has exactly the same symptoms as hemiplegia caused by lesion of the internal capsule in non-syphilitic individuals, as it is precisely the same arteries that are affected in both cases, although the arterial lesions may differ in pathological details. The monoplegia of intracranial syphilis is almost invariably due to a cortical lesion implicating one of the centres of the motor area. Paralysis of one or more of the cranial nerves is produced by a lesion at the base, and, as has already been said, this lesion is most frequently found over the optic, the olfactory, and the motor oculi nerves and the crura cerebri, although it may, of course, extend further back and implicate the other cranial nerves in the rear, such as the fourth, fifth, sixth, seventh, and eighth; and the symptoms will, therefore, be some impairment of vision, such as hemianopsia or blindness, paralysis of the motor oculi, occasionally of the olfactory nerves, and possibly of the intracranial nerves behind the third pair. With this paralysis of intracranial syphilis is frequently conjoined a hemiplegia due to implication of the crura cerebri, and as these crura cerebri contain the motor and sensory nerves that pass to the periphery of the opposite side, the hemiplegia will be upon the side opposite to the cranial nerves affected, so that there will be a crossed paralysis. Care must be taken, however, not to confound the optic neuritis that may result from a lesion at the base or from various other lesions of the cerebrum with the hemianopsia or blindness that is caused by direct implication of the optic tract. Syphilitic cerebellar disease is in no wise distinguishable from ordinary cerebellar disease.

Coma is occasionally observed in syphilis, extending over days or even weeks. It differs in no respect from the coma that may be produced by other intracranial lesions.

Epilepsy is due to some lesion of the cerebrum, and this may be either a general or focal lesion at the cortex, at the base, or in the substance of the cerebrum. In most cases it is due to a gumma. The epilepsy is sometimes accompanied by localizing symptoms which may enable us to judge of its site, or it may be without these, so that it may consist of a convulsion limited to one arm, one leg, or to certain groups of muscles, or it may affect both sides.

The most usual forms of insanity that are due, in my experience, to intracranial syphilis are dementia, confusional insanity, and hallucinations. The dementia is usually of the character of paralytic dementia. The patient is stupid, frequently with stupid non-logical delusions of a vague, indefinite character, and he may occasionally have the characteristic non-logical delusion of grandeur. The tongue and facial muscles are tremulous, the speech is tremulous and indistinct, there is the same characteristic defect in pronouncing certain

letters that is observed in paralytic dementia, and the gait gradually becomes shuffling, tripping, and uncertain. At times, too, the face flushes and the mental and physical symptoms become intensified, this exacerbation lasting for several days. The pupils frequently present the characteristic abnormalities of paralytic dementia. There is, therefore, no means of diagnosing this dementia from that of paralytic dementia except by the history of the earlier symptoms of the case or of a syphilitic infection. The French authors have called this form of dementia *pseudo-general paresis.* In other cases there is a simple dementia. The patient's expression is silly, he laughs without cause, he may lose his way in going out, cannot recall the names of friends and intimates, may have some suspicions of those about him, and altogether acts in a foolish and childish way. In some cases symptoms of confusional insanity will be present. The patient will be restless, at times excitable, muttering, confused, possessed of vague hallucinations, generally of hearing. Hallucinations are occasionally observed with but slight mental impairment. These are usually hallucinations of hearing, occasionally of smell.

Spinal Syphilis. Syphilis of the spinal cord, as has already been said, usually affects the membranes of the periphery of the cord, and only exceptionally extends into the spinal parenchyma, so that the clinical symptoms are generally those of a meningitis. Any portion of the cord may be affected, and the symptoms will, therefore, vary according to the locality. (*Vide* table on page 82.) Usually there is a bilateral paralysis of motion and sensation, with early exaggeration of the tendon-reflexes and contracture, and generally this is quickly followed by atrophy of the paralyzed muscles. If the dorsal cord is affected, there is apt to be impairment of the bladder and rectum and diminution or loss of sexual power.

Syphilis of the Peripheral Nerves. Syphilis affecting the peripheral nerves may cause neuralgia, or neuritis of the simple or multiple type. Syphilitic neuralgia or syphilitic simple neuritis are not generally distinguishable by themselves from the non-syphilitic forms, and care must be taken not to confound the nocturnal exacerbations of the simple form for the nocturnal exacerbations of syphilis. Syphilitic multiple neuritis is not as yet distinguishable by itself either from the non-syphilitic form, and, indeed, the only case of syphilitic multiple neuritis that has been reported is that of Taylor's, in which the symptoms were almost identical with those of Morvan's disease.

Besides these more usual forms of intracranial syphilis, spinal syphilis, and syphilis of the peripheral nerves, there may be different forms that it is impossible to classify, and of which the diagnosis can only be made by a knowledge of the earlier history or of the syphilitic infection. Thus, there may be lesions of single cranial nerves; there may be focal lesions in different parts of the cerebrum and the cerebellum, from arterial disease or from gummata; there may be mania or melancholia, or other forms of insanity; or there may be focal lesions in the spinal cord; or there may be a diffuse cerebrospinal syphilis, giving rise to a bewildering mixture of symptoms.

Diagnosis. No one should read this section upon diagnosis without having first perused the sections upon the pathology and the clinical symptoms, so that a clear idea may be obtained of the multiplicity of the symptoms and the most usual types into which this seeming complex is resolved.

Intracranial syphilis should be suspected in any case of a quasi-periodical headache returning at a certain time in the twenty-four hours, either at or toward night, less frequently in the afternoon or morning, conjoined with marked insomnia; and vigorous treatment should be begun at this period, for although, as we have already said, the headache and the insomnia will disappear upon the supervention of any paralytic or convulsive symptoms, yet these paralytic or convulsive symptoms may indicate an irremediable lesion, whilst the headache and the insomnia are of an earlier period at which the progress of the disease can almost invariably, if not invariably, be checked. A careful inquiry should always be made for this headache and insomnia, for its importance is seldom recognized by the patient, and he may therefore forget to mention it, or, if his memory has been impaired, he may have forgotten it; indeed, it is always well to remember that patients with intracranial syphilis, like those affected with other intracranial disorders, are often not aware of their defects of memory, so that a reliable history can only be obtained from some one who has been in constant contact with them, best of all a wife, a sister, or one of the female sex, for they are invariably far shrewder observers of symptoms than males. Any symptoms of lesion of the intracranial contents that has been preceded by this peculiar headache and insomnia should be suspected to be syphilitic. Aside from this, however, a lesion at the base of the brain, especially at that anterior portion in which lie the optic chiasm, the crura cerebri, the olfactory nerves, and the third pair, is usually either syphilitic or tuberculous. By this I do not mean to say that *all* lesions of this particular portion of the base are syphilitic or tuberculous, but simply that the proneness of syphilis and tuberculosis to affect this site should be borne in mind. Besides this, there are certain other groups of symptoms that should cause a suspicion of syphilis, namely: hemiplegia, under forty years of age, convulsions in an adult, and a comatose condition extending over days or weeks. Hemiplegia in an adult under forty years of age is suspicious because such a hemiplegia at this time of life is most likely to be caused by syphilitic endarteritis of the branches of the cerebral artery supplying the internal capsule. Hemiplegia, to be sure, occurs in the child from several causes unconnected with syphilis (*vide* "Cerebral Palsies of Childhood"); and also after forty from the endarterial changes ("Hemorrhages, Embolism, and Thrombosis"); but the age of the patient in the childish hemiplegia will make the differential diagnosis, whilst in the adult beyond forty there are frequently evidences of atheromatous arteries, a hypertrophied heart, and occasionally slight renal lesions indicative of an interstitial nephritis. Possible sources of emboli must be excluded, of course, in the consideration of such a hemiplegia, such as lesions of the heart, of the lungs, etc. Exclusion being made, how-

ever, of the hemiplegia of childhood, of that of degenerative endarteritis after middle age, and of cerebral embolism, the diagnosis will generally be that of syphilitic endarteritis. Syphilis should be suspected of being the cause of convulsions in the adult which have not been preceded by convulsions in infancy, and which are not of traumatic or nephritic origin, or due to pregnancy, or in an individual subject to migraine. It is too much the custom, I think, to regard convulsions in infancy as harmless and not prone to recur. I have elsewhere ("Epilepsy") called attention to the fact that a large proportion of the cases of epilepsy in youths or adults would be found on careful inquiry to have had a convulsion in early life that had usually been attributed to dentition or intestinal or febrile complaints; and of such early convulsions it may be very difficult to obtain a history unless the parents or some other near relatives are interrogated. The convulsions occurring in nephritis or in certain women during pregnancy are too well known to need more than a bare reference. Again, it is a curious fact, to which I have elsewhere referred ("Epilepsy"), that migraine is interconvertible in certain individuals with epilepsy, so that most persons who have a personal or hereditary history of migraine will be found, upon minute and patient interrogation, to have had a loss of consciousness or some epileptic-like convulsions at some period of their life. If, however, pregnancy, migraine, nephritis, trauma, and preceding infantile convulsions are excluded, a convulsion in an adult should give rise to grave suspicion of intracranial syphilis. Syphilis should also be suspected when there is a condition of coma extending over days or weeks, not traumatic, meningitic, diabetic, nephritic, or from typhoid fever. It is not always easy to exclude the coma of meningitis when the symptoms are of recent occurrence, and one must often wait for the characteristic retraction of the head or the temperature-curve. It should, moreover, be borne in mind that there may be a meningo-cerebritis of the middle portion of the ascending frontal convolution, just where the centre for the muscles of the neck has been located by Wernicke, giving rise to a retraction of the head as in ordinary meningitis, and that this symptom may be due to a localized meningitis possibly of syphilitic origin. Fränkel has reported such a case from the wards of the Charité Hospital in Berlin. The coma of nephritis, it must be borne in mind, is not always of the profound nature that is seen where the urine contains large amounts of albumin. It may be light and variable in its manifestations, simulating a profound hysterical condition. I have seen several such cases that were thought to be due to meningitis. Nor will it do to exclude nephritis by a mere cursory examination of one specimen of the urine for albumin. Several specimens should be obtained ; best one at morning, mid-day, and evening, and the amount of urea should be estimated ; casts, blood-corpuscles, and pus should be searched for, and albumin should be sought by the finer modern methods rather than by the old and rough test of heat and nitric acid. Diabetes can always be easily ascertained. Some anomalous cases of typhoid fever may mislead one at the start, although the progress of the disease almost invariably

makes the diagnosis very certain. We should therefore suspect intracranial syphilis if there are present

1. Quasi-periodical headache that returns at a certain time in the twenty-four hours, most frequently at or toward night, less frequently in the afternoon or morning;

2. Paralytic or convulsive symptoms that have been preceded by this characteristic headache and insomnia, when the headache and insomnia will have suddenly ceased upon the supervention of the paralysis or convulsion;

3. Symptoms indicative of a lesion at the base of the brain, preceded or not by the characteristic headache and insomnia;

4. Convulsions in the adult which have not been preceded by convulsions in infancy, and are not of traumatic or nephritic origin, or due to pregnancy, or in an individual subject to migraine;

5. Hemiplegia in an adult under forty years of age, even when there has been no preceding headache and insomnia;

6. A comatose condition extending over days or weeks, not traumatic, meningitic, diabetic, nephritic, or from typhoid fever.

If a history of syphilitic infection has been obtained, this will, of course, very materially assist the diagnosis, but it is frequently very difficult to obtain such a history. Women often do not know that they have been infected, any more than physicians who have become tainted at some surgical operation or examination. There are often many motives for concealment, because an acknowledgment of syphilis would materially affect the patient's interests, or his or her station in life. Then, too, a hard chancre may readily be overlooked because of the slight local symptoms, and this has occurred to some of the best syphilographers who have had the patient under constant observation from the supposed day of infection. In any case, therefore, where the above groups of symptoms are present, the patient should be put upon anti-syphilitic treatment.

The diagnosis of spinal syphilis is much more difficult than that of the intracranial affection, so that in the former case the diagnosis must rest mainly upon the history of syphilitic infection. Grave suspicion may, however, be entertained in any case of a spinal lesion in which there is a bilateral paralysis with early spastic symptoms and atrophy, and this suspicion will be strengthened when there is a spinal lesion of the dorsal cord with the above symptoms, and yet with no implication, or but slight implication, of the bladder and rectum. Tabes dorsalis has, undoubtedly, in a large number of cases a history of syphilitic infection, but yet it cannot be classified as a syphilitic lesion, and it really is only one of the syphilitic sequelæ.

The diagnosis of syphilis of the peripheral nerves must rest entirely upon the history of the syphilitic infection, as no characteristics of syphilitic peripheral nerve-lesions are known.

Diffuse cerebro-spinal syphilis may be diagnosed by the presence of characteristic intracranial symptoms, *plus* the symptoms of a spinal affection.

Prognosis. The prognosis of syphilis of the nervous system is largely dependent upon the question as to whether the lesions are

destructive or non-destructive. In general terms it may be said that the prognosis is good except where actual organic disease has been set up, or except where the syphilitic infiltration has excited in the normal tissue the formation of some other structure that is foreign to the particular texture. The infiltrations of the nervous tissues themselves, the gummata, the arterial lesions, are by themselves remarkably harmless. It is singular to see, as we often do, how much and how long the nervous structure will present evidences of syphilitic disease and yet recover entirely under adroit and vigorous treatment. Buzzard tells of an artery that had been almost occluded, tested by the sphygmograph, and yet it was again made patulous by proper treatment. To speak in general terms, it may be said that the symptoms of good omen are: nervous syphilis in its early stage; lesions at the base of the brain; spinal lesions without preceding intracranial syphilis, neuralgia, and the lighter forms of neuritis. Those of uncertain prognosis are: long-standing syphilis of the nervous system; such forms of mental trouble as are evidenced by slight confusion, hallucinations, and mania; spinal lesions with hemiplegia. Of decidedly bad omen are: locomotor ataxia; pseudo-dementia; nervous syphilis in persons whose general health is badly broken; relapses in spite of anti-syphilitic treatment; nervous syphilis in persons who bear the iodides badly; syphilis of the peripheral nerves that has existed for a long time, or that has caused pronounced trophic alterations.

Treatment. I have very pronounced ideas in regard to treatment. I know of no reason for dogmatism except considerable experience, and that is my excuse for my dogmatism. I have but little faith in mercury, and I never rely upon it alone. Again and again have I seen cases go halting along in the most uncertain manner under mercurial treatment, yet the symptoms have been smoothly, gradually, and certainly removed by the iodide. In several cases, too, I have known of death, although the most careful mercurial treatment had been pursued in enormous doses, and these cases have been precisely similar, as far as one is able to judge of one case by another in the practice of medicine, to others that I have been accustomed to regard as of favorable prognosis under treatment by the iodides. But the dose of the iodide which is administered by the Germans and French will often be entirely inadequate. I give the iodides until the symptoms yield or until iodism is produced. I make use of the saturated solution of the iodide of potash, each drop of which contains almost a grain of the salt. I commence with twenty drops three times a day, and increase it each day by two or three drops at each dose, and keep increasing until the symptoms yield or iodism is produced. Should iodism ensue before the symptoms yield, I pursue one of two methods. I first increase the dose of the iodide by about one-third, and rapidly increase each succeeding day. Singular to say, in some cases this increased dosage will cause the iodism to disappear, and the larger doses will be borne very well. Should, however, these larger doses still further increase the iodism, I decrease to one-half the dose at which the iodism had begun, and con-

tinue this decreased dose until the iodism diminishes or disappears, when I again rapidly increase the dose, and am usually able to go on without further trouble. In some few individuals, however, no amount of care will cause more than a certain amount of the iodide to be borne, and such cases, as I have said, are usually of unfavorable prognosis. In some cases—fortunately, they are rare—even small doses of the iodide will produce a cardiac disturbance that prohibits its administration. But this last fact should not be confounded with the phenomenon that the profession generally does not seem to be cognizant of, namely, that small doses of the iodide, 5 or 10 grains, will usually produce more iodism than the larger doses. I am perfectly well aware that the medical chemist will object that these large doses of iodide pass through the body and are quickly excreted by the urine; but I am equally well aware, in the face of this fact, that these large doses of the iodide will cause symptoms to yield that cannot be made to yield by less doses. I have given as much as 800 grains of the iodide of potash in twenty-four hours, and have seen symptoms disappear with these enormous doses that would not yield to minor ones. I administer the iodide after meals, and either in a full tumbler of ice-water, or in a glass of Vichy, or in a glass of a Bohemian spring water, the Giesshübler. I prefer the Giesshübler to the Vichy, although it is more expensive, because it is much more pleasant to the taste and agrees much better with the average stomach. Moreover, the Vichy water which is put up in siphons in this country is artificial, by no means equal in its effects to the natural water, and the natural water, either of this country or of Europe, does not keep so well in bottles as does the somewhat similar water of the Bohemian spring.

But he who expects to cure his patient with the iodides alone, unless the case be so grave as to call for immediate relief, will be disappointed. The human organism must be put in the best possible condition. A generous diet should be employed. Freedom from care and worry, where possible, should be enjoined, and all strenuous exertion of mind and body avoided. And let me say here that in many broken-down individuals the most satisfactory results may be obtained from a conjunction of the iodides with the brilliant treatment that has been made known by Dr. Weir Mitchell, under the name of "Fat- and Blood-making"—a therapeutical procedure that, in my humble opinion, will rank in coming years with the surgical revolution inaugurated by Lister. ("Neurasthenia," Chapter IX.) When the acute symptoms have been brought under control, travel —especially an ocean voyage—will often work wonders. In every case that tends to chronicity, staple tonics and alteratives should be used.

I have often asked myself, When can a person be considered to be cured of nervous syphilis? and I have never succeeded in answering the question to my own satisfaction. I know of no data by which we can guide ourselves. I have seen relapses occur years after the disappearance of every untoward symptom, and I have seen them even where a certain moderate treatment by the iodides had been

maintained. I usually impress this uncertainty upon my patients, warn them to be on the lookout for future symptoms, and apprise whoever may be their attending physician of their possible significance, and I enjoin them to keep up moderate doses of the iodide for years. This last precaution I observe the more readily because I have but seldom seen any ill effects from the continued administration of the iodides, to which patients usually become accustomed as they do to common salt. Occasionally it may happen that an obstinate pharyngeal or post-nasal trouble may be induced, or some unsightly cutaneous eruption; but, as a rule, these are controllable, and in any event they are far preferable to the possibilities of nervous syphilis.

DISSEMINATED SCLEROSIS.

Synonyms: Insular sclerosis. Multiple sclerosis. Sclérose en plaques desseminées. Multiple cerebro-spinal sclerosis.

History. Cruveilhier mentioned disseminated sclerosis for the first time in his celebrated *Atlas* in 1835. In 1838 it was also described by Carswell. Türck in 1855, Rokitansky in 1856, Frerichs and Valentina in the same year, Rindfleisch and Leyden in 1867, Zenker, Charcot, and Vulpian in 1862, have since dealt with thesubject, although it does not seem to have been known in England even in 1868. The lesion in children was only called attention to in later years, and the first case seems to have been that of Honemaker in 1879, although since then a number of cases have been published by Unger, Leube, Charcot, and others, and altogether the best *résumé* of the subject in children is that which has been written by Pritchard.

Pathology. Disseminated sclerosis consists, as the name indicates, of a number of insulated islets of sclerosis throughout the brain or the spinal cord, or both. These consist of circumscribed grayish spots, more or less regular in contour, sometimes discrete, sometimes confluent, whilst the surrounding nervous parenchyma has undergone no alteration. These sclerotic patches are found in the walls of the ventricles, in the white substance of the centrum ovale, in the septum lucidum, in the corpus callosum, in the optic thalami and corpus striatum, occasionally in the cerebellum, in the medulla oblongata, in the pons, and in the spinal cord. When first exposed to the air these grayish spots take on a roseate tint like that of the flesh of salmon. Even the nerves may be affected at their origin from the brain or spinal cord, and of the cranial nerves the optic, olfactory, and trigeminus are most prone to be implicated. In some cases the patches are raised above the surrounding surface and seem as if turgescent, although sometimes they cause no alteration in the superficial height of the nervous organ, and are even occasionally depressed. The consistence of these patches is usually somewhat firmer than that of the brain-tissue, and they are sometimes quite hard. In the brain the cortex is seldom affected, whilst in the spinal cord the patches appear upon the surface, and sometimes the whole

thickness of the cord is affected. Microscopically there is found to be a hypertrophied neuroglia, glia-cells, and fibrous spider-cells, and in the later stages a dense mass of fine fibres and also a certain number of fatty granules and globules, probably the remains of the degenerated nerve-element. The nerve-fibres are peculiarly affected; the medullary sheath undergoes degeneration, but the axis-cylinders persist to a remarkable degree, although they may ultimately disappear, whilst all the vessels are thickened, and occasionally the nerve-cells are atrophied. There is occasionally slight hypertrophy of the connective tissue of the pia mater, but this is rare. What the pathogenesis of the affection is we do not know, or whether it is a primary sclerotic disease or an outcome of vascular disease. Occasionally diffuse sclerosis is found in conjunction with the islets that have been described. I believe, however, that the symptoms of disseminated sclerosis can be caused by other lesions than those which have just been narrated, for in one case of mine which was so typical that I had lectured upon it for many years, and which I had in my hospital wards for three years under daily observation, there was found to be a meningitis of the pia mater, lepto-meningitis cerebralis, and in this case there was no history of syphilis, none of the cheesy nodules of syphilitic sclerosis, nor any of the other pathological lesions of sclerosis.

Clinical History. To anyone who has read this section upon the pathology of this disease, it will be apparent that there must be two sets of clinical symptoms, namely, general ones and localizing ones. In other words, there are certain symptoms common to disseminated sclerosis wherever the site of the patches may be, whilst there must be localizing symptoms due to impairment in function by the patches of some particular portion of the cerebro-spinal axis.

The general symptoms are:

Tremor;
Slight weakness or inco-ordination;
Nystagmus;
Increase of tendon-reflexes;
Contracture;
Optic-nerve atrophy;
Peculiar speech;
Vertigo.

The tremor is generally present only when voluntary movements are made, and to this the name of *intention-tremor* has been given. The best means of evoking this is to have the patient take a glass of water in his hand and carry it slowly to his lips, when the tremor will appear; or have him to write his name upon a blackboard, or take a moderately heavy book in his hand, or do any other acts that call for the exercise of volition. This tremor, therefore, disappears usually when the patient is at rest. It is a fine tremor, usually somewhat coarser than that of paralysis agitans, and in many cases becomes extreme and coarse when voluntary movement is made. Great stress has been laid upon this intention-tremor as a diagnostic means whereby we may distinguish disseminated sclerosis from paral-

ysis agitans, and I think that in the majority of cases this is a valid distinction, yet in some cases of paralysis agitans the same characteristics of tremor may be observed, whilst in some cases of disseminated sclerosis the tremor is also present when the patient is at rest, although this last is exceptional. With this tremor usually goes a slight inco-ordination, which is shown by the difficulty that the patient evinces in carrying a glass of water to the lips, or in writing, although it is impossible to say how much of this inco-ordination is due to the tremor and how much to actual inability, aside from the tremor, to make the different groups of muscles act together harmoniously. With the tremor and inco-ordination there is a slight paresis, which can be shown by the feeble grasp of the hand, either in pressing the physician's hand or in testing by the dynamometer, or in the feeble hold of a pencil or a piece of chalk. As in the case of the inco-ordination, however, it is difficult to say how much of this is from the tremor.

Nystagmus is a common symptom, and, like the tremor, it is usually present only on movement, consisting of movements of the globe in each direction, generally more marked in the lateral than in the upward movement of the eyes, or greatest when the eyes are directed to one side, but it is rarely rotatory. Occasionally a slight weakness in conjugate movements of the eye is also observed, generally to the sides. The tendon-reflexes are almost invariably increased, and this exaggeration is found in increased knee-jerk, ankle-clonus, and hasty micturition.

Contracture is a common symptom. In slight degrees it may only be evoked by the methods to which I have called attention on page 169, namely, extending the arm or leg, instructing the patient to let it go, testing by gentle movements of flexion and extension as to whether he has done this, then bringing the arm or leg to an extended position, and suddenly, without warning, flexing the limb, when, if there is any commencing contracture, a sudden click-like muscular resistance will be experienced by the hand before the flexion is completed. When contracture is more pronounced, a rod-like extension and a wax-like resistance that is offered to the hand in movements of flexion will make the symptom very evident.

Optic-nerve atrophy is a frequent symptom, either as the result of a primary atrophy or from a patch of sclerosis on the optic chiasm or in the optic nerve, and sometimes it is associated with impairment of vision or even segmental defects of vision.

The speech of disseminated sclerosis is characteristic. It is jerky and scanning or *staccato*, so that the patient hesitates; there are irregular, jerky movements about the muscles of the mouth and the tongue, and the teeth are uncovered in utterance. In my opinion, the speech is of more value as a means of differential diagnosis from paralysis agitans than the tremor, because in shaking palsy the speech is the very antithesis of the speech in disseminated sclerosis, being slow, deliberate, seemingly wise.

Vertigo is a frequent symptom, although it is not in my experience

so common as Charcot would have it to be, and it is rarely accompanied by vomiting.

These are the general symptoms.

In addition to these there may be localizing ones of great variety due to the implication of some part of the brain, cerebellum, spinal cord, or peripheral nerves by a sclerotic patch. An attempt has been made to distinguish between the cerebral symptoms and the spinal ones, but this can only be done by means of the localizing signs and not by means of the general ones. It is useless to attempt to catalogue the different localizing symptoms, because they are as various as are the functions of the nervous system. None of the general symptoms have been explained by the localizing of patches, with the possible exception of the optic-nerve atrophy. In general terms, it may be said that if a sclerotic patch implicates the motor tract, it will cause paralysis. An increase in the tendon-reflexes and contracture, it must be borne in mind, however, is not evidence of an implication of the lateral columns, as some observers have stated, for these symptoms are found when the lateral columns are not affected. In very few cases, as we have stated, is the gray matter of the cortex affected, so that when there are lesions referable to this gray matter, such as dementia, as a rule disseminated sclerosis should be excluded except in children. If the cerebellum is affected, the characteristic symptoms of cerebellar disease will be added to the general symptoms. If there is a sclerotic patch destroying the functions of certain spinal areas, symptoms of impairment of such areas will be present, and the most frequent of these seems to be some of the symptoms of locomotor ataxia, such as loss of the knee-jerk and marked ataxia, which are probably due to the implication of the posterior columns by a sclerotic islet. The duration of the disease is indefinite. Thus, I have known one case to last fifteen years, and another to terminate in two years, whilst Gowers speaks of one that lasted only a year and three-quarters. The disease may gradually progress to great helplessness without actual paralysis, or there may be a sudden apoplectoid attack, or the patient may die of failure of respiration. The nutrition is usually unimpaired throughout. When the disease occurs in children, it has certain peculiarities that are not so often present in the adult. Thus, the sudden onset is most common, and this may succeed a convulsion. Strabismus is frequently an early symptom. A low standard of intelligence is the rule. Convulsions were observed in more than half the cases. Contracture is not nearly so frequent as in the adult. Tremor and ataxia, however, are quite as constant in the child as they are in the adult.

Etiology. The etiological factors which bear a certain relationship to disseminated sclerosis are:

Age;
Sex;
Heredity;
Exposure;
Emotions;
Trauma;
Febrile disease.

Most cases are found between the ages of twenty and thirty-five years, but the disease also occurs in children, and Dr. W. B. Pritchard, chief of my clinic at the New York Polyclinic, has collected from the literature more than fifty cases in children, ranging from fourteen months to fourteen and a half years.

The disease appears to be much more common among adult females than males, although in children the sexes are about equally divided.

Heredity is a frequent cause. The most remarkable proof of this was observed by Pelizæus, who observed the transmission of the disease through successive generations, the symptoms manifesting themselves in the male members of the family only; but instead of the direct heredity of disseminated sclerosis, there may be simply a general neurotic heredity.

Exposure to cold, and especially to damp cold, has frequently seemed to be a cause.

The emotions are potent factors, as in paralysis agitans; thus Spitzka saw a case develop from fright in a cigarmaker, and Wilson narrates the history of a child one year old who was thrown into a convulsion by having a goose thrown at her, and after the convulsion the disease developed.

Trauma is a frequent cause also.

Febrile disease, such as scarlet fever, measles, pertussis, diphtheria, smallpox, and typhoid are frequent causes in childhood, and Sparks has even seen a case developing after herpes zoster.

PROGNOSIS. The prognosis of disseminated sclerosis is hopeless. I am sorry to say that no case of cure has ever been recorded.

DIAGNOSIS. The diagnosis should be from:

Paralysis agitans;
Chorea;
Hysteria;
The metallic tremors and alcoholism;
Lepto-meningitis.

From paralysis agitans the diagnosis in the typical cases can be made with ease. This will be seen by the following parallel progress:

Paralysis Agitans.	*Disseminated Sclerosis.*
Age over forty.	Age under thirty-five, or even in a child.
Tremor usually present only upon voluntary movement.	Tremor usually present both upon movement and when the patient is at rest.
Speech slow, deliberate, wise, monotonous.	Speech jerky, tremulous, scanning.
Attitude characteristic: head bent somewhat forward, shoulders held stiffly; walk shuffling and stiff.	No attitude at all.
Bread-crumbling position of the fingers.	No special position of hand.
Nystagmus rare.	Nystagmus frequent.
Optic-nerve atrophy rare.	Optic-nerve atrophy frequent.
Impulse to go forward, or go faster and faster forward, or to go backward or laterally (propulsion, festination, retropulsion, lateropulsion).	No impulse to go in any direction.
Exaggeration of tendon-reflexes and contracture rare.	Exaggeration of tendon-reflexes and contracture frequent.

These signs in the two diseases will make, as I have said, the diagnosis easy in a typical case; but in a non-typical case, or in a case that stands at the borderland between the age of thirty-five and forty, the diagnosis may be very difficult indeed.

In the ordinary cases of Sydenham's chorea the diagnosis should not be at all difficult, because there is no tremor, and the muscular movements are jerky, quickly beginning and quickly ending, or wave-like in the athetoid variety. In some late cases of chorea, however, that have resulted from improper treatment of the original attack or the relapses, there may be a mixture of choreic movements and muscular spasm (not contracture) that may at first sight cause confusion, but the lack of true tremor and the other symptoms of the disease will soon make the matter clear.

In some cases of hysteria, especially if it be associated with tremor and hysterical paralysis of slight degree, there may be some confusion at first, but the history of the case and the lack of the many symptoms of disseminated sclerosis will afterward prevent continued confusion upon the subject.

Metallic tremors of lead, copper, and mercury are entirely lacking in the characteristic symptoms of disseminated sclerosis, so that the mere mention of this fact should prevent error of diagnosis.

In alcoholism the history of continuous drinking or of a recent debauch, and the absence of nystagmus, intention-tremor, exaggerated knee-jerk, optic-nerve atrophy, etc., are sufficient.

Lepto-meningitis cerebri—that is, meningitis of the cerebral pia mater—is, as I have seen in one case of my own which I have already cited, quite capable of causing the typical symptoms of disseminated sclerosis. In my case, however, there was a marked dementia that supervened within a year after the onset of the disease, and if this should prove to be true of cases that may be observed in the future, it will be a valuable means of differential diagnosis.

Treatment. I am sorry to say that the treatment of disseminated sclerosis amounts to nothing. I have never yet succeeded in doing more than causing some palliation of the tremor. For this purpose I find the bromide of potash and hyoscyamine the best drugs. The bromide of potash should be given in doses of 10 grains twice or three times a day, in conjunction with $\frac{1}{100}$ grain of the hyoscyamine of the crystallized form once or twice a day, as may be found necessary to mitigate the tremor. For the other symptoms I have never found anything of value, although I have patiently tried galvanism, faradism, ergot, iodide of potash, mercury, nitrate of silver, and hydrotherapy.

LEAD-POISONING.

Lead can be carried into the human body in many ways—by means of the mouth, the skin, and the lungs; and when taken into the body it can be stored up in surprisingly large quantities, so that as much as two grains have been found in the brain in a fatal case. The sources of poisoning are innumerable. The workers in lead-factories are most frequently affected, as well as many other artisans, such as painters, plumbers, type-setters, type-founders, glaziers, glass-grinders, etc. The occasional sources of lead-poisoning are of late years not nearly so numerous as they were a decade or more ago, but,

nevertheless they occur. Thus, preserved fruits, vegetables, acid fruits cooked in glazed earthenware vessels, shot left in wine bottles after cleaning, snuff packed in lead paper, hair-dyes, cosmetics, and flour ground in defective mills sometimes still induce the toxic effects of lead. From Marshall's experiments, it is probable that the absorption of lead is gastric, and not intestinal; but the most exhaustive series of experiments upon the effects of lead have been those of Prevost and Binet. Beside the clinical symptoms which are familiar to most observers, they have shown that there are diminution and alteration of the red blood-globules without increase in the white corpuscles, albuminuria, fatty degeneration, occasional pericarditis, with at times granulo-fatty changes in the myocardium and segmental degeneration of the peripheral nerves. Investigation has shown that the lead accumulated in the muscles and in the kidneys, and that this was always in proportion to the length of time during which the animal had been taking lead. It was also found abundantly as phosphate in the bones, the relative proportions of the mineral and organic constituents of the bone remaining unaltered. The liver generally contained but little lead in cases of prolonged administration, although the quantity might be increased temporarily after each dose; but it never accumulated in the liver as it did in the kidneys. Only small quantities were found in the muscles, spleen, nerve-centres, eyes, lungs, heart, pancreas, genital organs, and blood. Blyth, examining portions of the bodies of two persons dying suddenly from lead-poisoning, extracted from the liver $\frac{1}{3}$ grain of sulphate of lead, from one kidney about $\frac{1}{13}$ grain, from a cerebrum about a grain and a half, and from a cerebellum about $\frac{1}{4}$ grain. Putnam has examined the urine of eighty-six individuals, both healthy and suffering from lead-poisoning, and has arrived at some rather remarkable conclusions. He finds that the urine of persons in perfect health is almost free from lead; but that in neurasthenia and epilepsy a small minority have lead in the urine; that the lead increased in proportion as these nervous symptoms were associated with organic disease; that among the cases with organic disease lead was found most constantly in chronically diseased teeth, extending generally along a row, but sometimes limited to individual teeth; that those having least cachexia were those in which lead was not present; but that none was found in progressive muscular atrophy.

The symptoms of lead-poisoning are the so-called cachexia, the so-called lead-line on the gums, colic, pains, paralysis, cerebral symptoms, hemiplegia, and hemianæsthesia.

The so-called lead-cachexia consists of anæmia, due to actual diminution in the principal blood-corpuscles, as well as to impairment of muscular strength. Fever is very seldom present, and only in slight degree. The lead-line is an irregular bluish-black line along the gums at the junction with the teeth. Lead colic is too well known to need more than a bare mention. It is accompanied by vomiting, slight changes in the pulse, and constipation. The pains are either vague or referable to the muscles of the joints, sometimes conjoined with tenderness of the muscles, with slight sensory symptoms, such

as tingling, very rarely with anæsthesia. Motor paralysis, with atrophy, is a most frequent symptom in the case presented at our clinics. Thus, in 75 cases observed at the New York Polyclinic, this muscular paralysis and atrophy were present 69 times. The typical paralysis is that known as "wrist-drop." It is due to an inability to extend the fingers, sometimes all the fingers, sometimes only the two middle ones, or the first and second. The long extensors are the muscles primarily affected. But at this time, it should be remembered, the distal phalanges can be extended, as Duchenne has shown, by the interossei and lumbricales. Extension of the thumb is also generally impaired or lost, although its metacarpal bone can be extended. The extensors of the wrist next become

FIG. 160.

Case of lead-paralysis affecting the extensor muscles.

affected, and the wrist-drop then becomes complete. These different muscles are affected in varying degrees in different cases, sometimes first upon the radial, sometimes upon the ulnar side. As Duchenne has shown, the wrist can nevertheless in some cases at this period be extended when the fingers are strongly flexed, which is due to the fact that the extensor carpi ulnaris or radialis is unaffected. The affected muscles, therefore, are in a range of distribution of the musculo-spiral nerve, but the supinator longus, supplied by the same nerve, generally is not impaired, nor is the extensor of the metacarpal bone of the thumb. The paralyzed muscles are usually atrophied, the atrophy sometimes coming first, but generally the paralysis. These affected muscles are usually altered in their muscular reaction. There is sometimes the ordinary degeneration of reaction, sometimes merely a slight diminution in response to faradism. Erb and Gowers have seen a slight reaction of degeneration preceding the onset of the paralysis, the positive closing contraction

being more marked than the negative closing, and a continuous tetanic contraction being produced during the passage of the current. I have never seen this. Besides these muscles of the lower arm, some of those of the upper arm and shoulder are occasionally implicated, most frequently the deltoid, sometimes the biceps, brachialis anticus, and triceps. Paralysis of the legs is rare, but I have observed it in several instances, and the muscles affected are the peronei, supplied by the peroneal nerve, and the long extensors of the toes. In some rare cases there is a general paralysis. In the cases in which the atrophy precedes the motor paralysis, there is often a very close simulation of progressive muscular atrophy, more especially as fibrillary twitchings are found. Muscular cramps occasionally occur, generally in the legs. Tremor is sometimes also observed. Lead-poisoning sometimes has a purely local effect, causing impairment of sensation and occasional paralysis. The cerebral disturbances consist of convulsions, delirium, coma, hemianæsthesia, hemiplegia, and chronic mental disturbances, the latter most commonly taking the form of melancholia, sometimes simulating general paresis. These cerebral symptoms, however, are exceptional. When such general symptoms as convulsions, delirium, and coma are observed, their onset may be acute or preceded by such prodromata as vertigo, tinnitus aurium, and sleeplessness. The convulsions may be epileptic in form, or of the types of hystero-epilepsy or catalepsy. When there are delirium and convulsions, a condition of coma usually supervenes, but, although deep, this coma is not complete. A rise of temperature from 100° to $101\frac{1}{2}$°, or even 102°, is sometimes observed in conjunction with this symptom, and death may occur. Hemiplegia and hemianæsthesia are usually transient. The mental disturbances that have been mentioned are chronic. Ocular symptoms are occasionally observed in lead-poisoning, such as inequality of the pupil, amblyopia, bilateral amaurosis, or the ocular affections which usually go with hemianæsthesia, such as concentric limitation of the field of vision and loss of color-sense. Optic neuritis is by no means infrequent in cases of acute lead-poisoning, and when there is a cerebral implication there may be a simple atrophy.

The most frequent pathological lesions found in cases of lead-poisoning are those of neuritis, especially in the intramuscular nerve-fibres, although the changes are also found in the nerve-trunks. In some cases the neuritis begins in the medullary sheath; in others there is the ordinary degeneration of the nerve-fibres, such as is seen after section of the nerve; and in still other cases the neuritis affects the nerve-segments, which are separated by normal portions of nerve. Changes will appear in the peripheral portions of the nerve external to the anterior roots, although occasionally the latter are affected. The posterior roots are almost invariably normal The fibres of the atrophied muscles present the usual appearance of muscular atrophy. As a rule, the spinal cord and brain are unaffected, but in some cases the cells of the anterior horns have been atrophied, or spots of softening have been found in the anterior horns and the surrounding brain-matter, or there has been a thickening of the vessels of the

cord or a proliferation of the connective tissue, whilst in the brain slight meningitis (especially of the pia mater) and hyperæmia of the dura mater have been found.

The diagnosis of cases of lead-poisoning can usually be made with ease. The characteristic lead-line around the gums, the wrist-drop, the lead-colic, and the history of the exposure to lead will usually suffice. It must, however, be remembered that wrist-drop may occur from arsenical poisoning and from the effects of silver. In cases of arsenical poisoning, however, there is often an ataxia with impairment of sensation, and in cases of poisoning by silver there is the characteristic discoloration of the skin, and almost invariably the poisoning has been because of the medicinal use of the metal. If there is no history of exposure to lead, however, the diagnosis may be a matter of some difficulty, but a rigid investigation of the patient's environment will make the case plain. When there is simulation of progressive muscular atrophy from lead-poisoning there may be considerable trouble in reaching the correct diagnosis. In all doubtful cases examination of the drinking-water and of the urine should be made.

The prognosis of the nervous symptoms of lead-poisoning are generally good. All the cases that come to my clinic recover, and I have never seen cases in private practice that did not do the same.

The treatment of lead-poisoning should be by means of the iodide of potassium, salines, tonics, electricity, and rest in the severe cases. The iodide of potassium, in doses of 10 to 15 grains three times a day, is an invaluable drug in increasing the excretion of lead by the urine in some unknown combination. Some authors warn us against using the iodide in large doses when there are acute symptoms, in the fear that the toxic effects of the lead should be increased by its sudden passage from the tissues and into the blood. I have never seen any such result in an experience of over twenty years. The best saline is the sulphate of magnesia, and this should be used to produce one or two free movements, and then only as a laxative. It should never, however, be used to the point of depressing or weakening the patient, as is too often done. It should, moreover, be remembered that the excretion of the lead is not the only object of treatment, but that the human organism, weakened by its presence, must be sustained. In all cases, therefore, tonics should be made use of for a few weeks, and the best of these is iron, preferably the dialyzed form, the albuminate in doses of one drachm three times a day in a cup of water, or the peptonate, in the form of compressed tablet, three to five grains three times a day. The iron can usually be advantageously given with tonic doses of quinine, two grains three times a day, or the elixir of calisaya. In cases of colic, morphia should be used hypodermically to relieve the pain, and usually the morphia will act best when combined with atropia. The galvanic or faradic current is of invaluable service in treating paralysis, and should be applied to the affected nerves in the manner indicated upon pages 123 *et seq.*

CHAPTER IX.

THE NEUROSES.

EPILEPSY.

Synonyms: Latin, Morbus sacer seu comitialis. French, l'Epilepsie. German, Fallsucht. Spanish, Mal caduco.

DEFINITION. The term epilepsy, from ἐπιλαμβάνω, "to seize upon," means convulsions that are either tonic or clonic, of one limb or of several or of the whole body, with or without loss of consciousness; or loss of consciousness of a certain character; or certain phenomena that are supposed by some authors to be the equivalents of the convulsive or comatose symptoms.

HISTORY. Epilepsy was known as far back as the time of Hippocrates, but it was ill defined, and the knowledge of the clinical symptoms and the nature of the various forms has been the work of the writings of the last twenty years, largely synchronous with the great development that has taken place in this time in our knowledge of cerebral localization and the diseases of the brain, cerebellum, spinal cord, and peripheral nervous system.

CLINICAL SYMPTOMS. The main types of epilepsy are—

Grand mal;
Petit mal;
Convulsive movements without loss of consciousness;
Vertigo;
Double consciousness;
Hystero-epilepsy;
Procursive epilepsy;
Laryngeal epilepsy.

The symptoms of the typical attack of epilepsy are a sudden loss of consciousness, a sharp automatic cry, a fall, a series of tonic muscular movements of short range which are quickly succeeded by general clonic convulsions, these latter lasting for several seconds or minutes, when the patient becomes partially conscious, being half-dazed, and then gradually merges into what seems like a deep sleep, but is really a return to unconsciousness, this quasi-slumber being generally an hour or more in duration. This type is known as *grand mal,* or major epilepsy. The deviations from it, however, are innumerable. The most frequent non-typical group of phenomena consists of a loss of consciousness, either without convulsive movements, or with very slight ones. This is known as *petit mal,* or minor epilepsy. Or there may be convulsive movements of the fingers alone, particularly where the heredity is dying out in a family; or there may be a sensation of vertigo, which, indeed, very frequently occurs

in the individuals between the *grand mal* and *petit mal* attacks; or there may be other sensations which are called *auræ;* or there may be conditions of double consciousness, as it is called, in one of which a person may lead his usual life and in the other lead a totally different one. The most frequent of all these is either the *grand mal* or the *petit mal.* The color of the face in these attacks is very variable, being sometimes pale and sometimes suffused. It should always be remembered that statements regarding this color of the face are very unreliable, as the onlookers are generally too much disturbed emotionally to be accurate observers. The physician himself seldom sees an attack, even if he be the resident of a hospital; thus, while it would tax my memory for me to recall the thousands of cases I have treated in twenty years of professional life, I could easily count up all those that I have observed during the attack itself.

In some cases there is a queer combination of hysteria and epileptoid symptoms, varying from the pronounced hysterical attack to the frightful cases which are illustrated in the writings given out from the great Parisian hospital of La Salpêtrière; but these are seldom, if ever, seen in this country, and what we American physicians call hystero-epilepsy, and what the English physicians designate by the same name, is of a very much less sensational nature than what the French describe. True hystero-epilepsy rarely begins with a cry or change of color in the face, but generally with the tonic convulsive movements of true epilepsy. These are followed by the period of so-called "clownishness," characterized by all sorts of *bizarre* movements, opisthotonus, distended abdomen, grimaces, etc. Then succeed a series of passionate attitudes, with delirium. It is claimed, too, that ovarian compression will cause the symptoms to cease, but this has not been so generally in the cases which I have seen. All these types of hystero-epilepsy, however, are different from the true epilepsy in the admixture or predominance of purposive or seemingly purposive movements instead of a rigidly tonic or clonic movement of a shock-like character, whilst opisthotonus, a non-epileptic symptom, is occasionally observed in them.

Procursive epilepsy has been recently described by Mairet, and consists of running movements forward, but otherwise presenting the usual phenomena. It may alternate with ordinary epilepsy, or precede or merge into it.

The so-called laryngeal vertigo is unquestionably a variety of epilepsy, and the term vertigo should be discarded, because, as is stated under "Vertigo," this latter term should be reserved for subjective sensations of loss of equilibrium, with either no impairment of consciousness or very slight implication of it. Laryngeal epilepsy has not as yet been described in children, but it probably will be in the course of time. It consists of a slight loss of consciousness, occasionally with light tonic or clonic movements, and the exciting cause is some laryngeal, tracheal, or bronchial affection, such as laryngeal tumors, asthma, etc. Those cases which have been reported so far have ceased after the cure or removal of the seeming cause,

but the histories have not been extended over a sufficient length of time to exclude the possibility of recurrence.

Certain dream-like states may precede the typical manifestations of epilepsy, or take their place. They are simply degrees of impairment of consciousness. Of the latter, the condition of double consciousness is the most marked. One patient of mine, a lad of fourteen, would wander for days about the lower quarters of New York, sometimes earning wages in some light occupation, without arousing the faintest suspicion in those about him, until some fine morning he would return to his normal condition and then quietly wend his way home.

Falret has spoken of another equivalent of epilepsy which he calls the larvated epilepsy (*epilepsie larvé*). This may consist of an outbreak of hysterical insanity, usually of mania, or, as I have occasionally seen it, an attack of violent migraine or of great irritability of temper. Falret claims that in these cases there is never any petit mal. Hughlings-Jackson, however, calls attention to the fact that many cases of petit mal are so slight, consisting only of a very momentary impairment of consciousness, with some slight muscular twitchings about the face and eyelids or even of the fingers, that they may readily be overlooked by the friends of the patient, whilst the impairment of consciousness, however slight, prevents the patient from himself recognizing them, so that any symptoms following these might be thought to be actual equivalents, whilst they are really attacks of petit mal succeeded by the phenomena that have been mentioned.

The epileptic insanities are treated in another chapter of this book.

Our conceptions of epilepsy will be greatly simplified if, at the outset, we recognize the indubitable fact that epilepsy is but a symptom, just as is a cough or fever, so that epilepsies may be divided into those that are due to recognizable organic disease, those that are reflex, and those that we may call idiopathic.

The clinical characteristics of the idiopathic form of the ordinary grand mal or petit mal will occupy us for the present. This has certain features which are of great importance from a therapeutic and occasionally from a diagnostic standpoint, and salient among these characteristics are the nocturnal recurrence of some, the quasi-periodicity of others, the association with migraine, and the temporary response, usually in a favorable way, to slight changes in the environment or the treatment. The nocturnal recurrence of epilepsy may, however, readily be overlooked, and should always be suspected in a child who is hereditarily predisposed to the malady or who wakes in the morning exhausted and pale. Some eleven years ago I first called attention to the quasi-periodicity of many epilepsies. Many epileptics will have a recurrence of their attacks at certain times. Thus, females are more especially subject to it at or about the menstrual period, while in others it will recur at certain times of the month or at certain intervals, these periods of relief sometimes lasting for months, and in some rare cases even years. I shall indicate, in the proper place, how this quasi-periodicity may be utilized for

therapeutic purposes. Some eleven years ago I called attention to the association of epilepsy with migraine, not knowing until some time after the publication of Fére's book in 1890 that he had done me the honor to reproduce the history of my case in that great work upon epilepsy, and that Tissot, Parry, and Living had previously observed the same association. In these cases epilepsy alternates with migraine, the migraine disappearing when the epilepsy appears, and the epilepsy returning when the migraine disappears. By this I do not mean to say that all cases of migraine are subject to epilepsy, but I do mean to say that there is a very close relationship between migraine and epilepsy, and in some cases the relation is so close as to permit of this alternation; indeed, almost all cases of migraine will be found at some period of their lives to have had a loss of consciousness, with or without convulsive movements, although generally this fact is strenuously denied. But this association of migraine and epilepsy is of considerable therapeutic significance, as we shall see. Epileptics, as is not generally known, are very readily influenced by slight changes in the environment and in the treatment. This was first observed as far back as 1828 by the great Esquirol, at the head of the Salpêtrière, who divided his epileptics into groups, putting each group upon a different medicine, and one group upon a disguised placebo; they all improved for a certain length of time, they all relapsed at about the same time, and each medicament had as much effect as the placebo, and no more. In addition to this, I have pointed out again and again, until my neurological brethren in New York now twit me for my persistency in calling attention to the matter, that epileptics will do well for a time upon any change of treatment, whether that treatment be medical or surgical, whether it consist of cutting off the prepuce, removing the clitoris, extirpating the ovaries, doing operations upon the male or female genitalia, using the hot iron or moxa, cutting the eye-muscles, or even, as I have done myself, etherizing the patient and cutting a piece of skin out of the buttock. I have even seen improvement effected in a patient for months by mere change of locality, as in one patient of mine coming from a distant city in Illinois, who had no attack for eight weeks while she was in this city, although she had been having three or four paroxysms in a day before coming here, had the same number when she went back home, and received nothing but a few placebos from me whilst here. I know of another inveterate epileptic whose fits were banished for months after she had fallen down a flight of stairs into a vat of hot water, not returning until the process of cicatrization was completed. These epileptic attacks are very prone to recur in variable spells—either every day, often several in a day for weeks, perhaps months, and then spontaneously disappearing for a different length of time, to recur again and again in the same old manner. I point this out in almost every epileptic that I lecture upon at the New York Polyclinic.

Most epileptics, as I have shown, have large and variable pupils. Marie and Musso have attempted to show by measurements of the pupil that my statements upon this point were erroneous, but the

very variability of the pupils renders their measurements questionable. By this I do not mean to say that the pupils are not large and mobile in other diseases, but simply desire to call attention to the fact that this peculiarity does exist in epilepsy. A trained eye can readily detect the *facies* in most epileptics, although it is impossible to describe its fleeting traits and photography is not yet far enough advanced to portray it, probably because the expression is most marked when the patient is not directing his attention to any one point, so that when the muscles of the face are on the alert, so to speak, in the taking of a photograph, this peculiar expression disappears. I think, however, that I could make a diagnosis of epilepsy in most cases by the *facies* alone. In the inveterate cases which have been for a long time under treatment by the bromides it is much more marked, of course, and at this period the accompanying restlessness, the pallor, the acne, the coated tongue, and the peculiar breath will greatly aid in the diagnosis. The temper of most epileptics is extremely irritable, and especially is this the case in children whose malady has procured for them ill-advised license.

The epilepsies of organic origin or of probable organic origin are as variable as are the functions of the brain. Hughlings-Jackson first called attention to a variety in which the convulsive movements were localized in one or more limbs, and this has come to be known as Jacksonian epilepsy. It has been found in many instances to be due to recognizable gross organic disease of the cortical motor centres or the underlying motor tract. Procursive epilepsy is believed by Mairet to be due—upon what grounds we shall learn later on—to cerebellar lesion. Epilepsy may be found among the symptoms of almost any disease of the brain or spinal cord, and it may also be present with tumors of the brain or cord, the different forms of cerebral and spinal meningitis, hemorrhages of the brain, traumata of the brain and cord, etc.

Etiology. The following factors seem to bear some relationship to epilepsy:

Age;
Sex;
Heredity;
Migraine;
Organic brain, spinal, or peripheral lesions;
Traumata;
Lesions or impairment of function of non-nervous organs;
Hysteria;
Malnutrition.

Gowers has analyzed 1450 cases as follows:

Under 10 years	422 cases.
From 10 to 19 years	665 "
" 20 " 29 "	224 "
" 30 " 39 "	87 "
" 40 " 49 "	31 "
" 50 " 59 "	16 "
" 60 " 69 "	4 "
" 70 " 79 "	1 case.

It is not generally known that, as I have been for years in the habit of pointing out in my lectures, many cases of idiopathic epilepsy will begin with a fit in early infancy, after which no convulsion may occur in a year or years, when the attacks will again appear, and at this or some later period the case will become one of well-marked epilepsy.

In Gowers's 1450 cases the percentage of females was 54.6, and males 46.4. There was a hereditary history in 36.6 per cent.

The association of migraine with epilepsy has already been spoken of, as well as the connection with organic brain and spinal lesions and traumata.

In some rare cases epilepsy may be caused by diseases of the heart and of the kidneys, occasionally by impairment of function of the gastro-intestinal organs, rarely of the lungs, and very rarely, indeed, if ever, of the genitalia.

The association of hysteria and epilepsy is by no means frequent.

A fact of immense practical importance, and one that seems to have been almost universally overlooked, is that profound malnutrition may be a cause of epilepsy, so that with the restoration of the general health the epilepsy will disappear. In one case of mine a cure was maintained for nine years, in another for eight, in another for four, in another for three, and in another for five, and in none of these was there any hereditary predisposition.

Pathological Anatomy. Epilepsy, as I have said, is merely a symptom, and in every case it is a question as to what it is a symptom of. From a pathological standpoint we may therefore divide epilepsy into two great classes, namely: epilepsy from recognizable organic disease; second, epilepsy which we may call idiopathic.

The organic lesions causative of epilepsy are: tumors of the brain and cord; meningitis, either tubercular, cerebro-spinal, suppurative, or from disease of the ear, or by metastasis from other organs; the cerebral palsies of childhood, such as hemiplegia, double hemiplegia (diplegia), or paraplegia, and due to such lesions as porencephalitis, embolism or hemorrhage from the cerebral arteries, thrombosis of the cerebral arteries or veins; lesions of the cerebellum; sclerosis of the brain and cord, either of the type of multiple sclerosis or of other forms; syphilis of the nervous system; and diseases of the peripheral nerves.

We know very little about the pathology of the idiopathic forms. The old theory of vasomotor spasm producing cerebral anæmia or hyperæmia is now almost entirely discarded by neurologists, as it ought to have been long ago, for there has absolutely never been any logical proof of it whatsoever. It is undoubtedly true that ligature of the carotid artery and great hemorrhage are capable of producing epilepsy; but it has never been demonstrated that the profound ischæmia—not anæmia, as it is so often incorrectly termed—thus produced was present in every case of epilepsy, and on the contrary there are many experiments and facts going to show that the cerebrum of epileptic human beings and animals is not vascularly

altered to any marked degree. Vulpian, for example, has examined the brains of guinea-pigs artificially rendered epileptic, and has found that they are neither hyperæmic nor ischæmic during an attack. The deductions as to the cerebral circulation that are drawn by certain authors from the facial circulation are unwarrantable, as the latter is by no means an index to the former, for it often happens, especially in cerebral traumata, that the face may be deathly pale at the same time that the cerebral meninges are in a condition of profound hyperæmia. The same remark also applies to the proof that would be drawn from the condition of circulation in the retina. In some cases of idiopathic epilepsy a sclerotic condition will be found in the brain; in others an old focal meningitis, especially in the pia mater; whilst in many no lesions at all are visible. Sclerosis of the cornu ammonis as a probable cause of epilepsy has caused a great diarrhœa of words, but it is by no means constantly found, and even when it has been, it has never been shown whether it was a coincidence or an effect rather than a cause. Barthez and Rilliet have laid great stress upon a peculiar variety of sclerosis to which they have given the name of tuberous or hypertrophic, and in which the convolutions are found elevated, studded with round or ovoid tuberosities irregularly disposed upon the convex portions of the convolutions, rarely in the fissures, the size varying, but sometimes attaining that of a large nut. These nuclei do not affect the white matter, the gray matter of the cortex, or the central nuclei, the basal ganglia being alone affected. But this variety of sclerosis is open to the same objections as those which have been dwelt upon in regard to the sclerosis of the cornu ammonis. Chaslin examined five epileptic brains at the instance of Féré, and found what he called neurogliar sclerosis in four of them. In this condition the convolutions are macroscopically shrivelled, small, hard, smooth, or slightly roughened, the pia mater not being adherent, or otherwise abnormal, the pathological alteration extending in a very variable manner over the surface of the cerebrum with large intervening normal portions sometimes reaching to the medulla oblongata or to the cornu ammonis. In only one case was one of the olivary bodies seen to be thus affected. A microscopical examination of the fundamental lesion has demonstrated that it is essentially due to a number of rough fibrillæ of an uncertain length which had invaded the cerebral tissue, especially the gray cortex. In the author's own words, "In the normal state the first layer of the gray cortex contains certain so-called spider-cells, whose prolongations are scarcely visible. In this condition, on the contrary, this first layer is formed by a bundle of fibrils arranged nearly parallel to the cerebrum, and it can be distinctly seen to originate from numerous cells with hypertrophied prolongations. In the preparation which I am viewing at this moment there is a place where this transformation invades all the layers, but leaves intact numerous nerve-cells and vessels. It can be seen, moreover, that these fibrillæ in a certain space form in the depth of the cortex a network of nodal points, in which lie the cells of the neuroglia. Finally, and I would call attention particularly to this fact, this first layer is

studded in places by large compact bundles, which are evidently formed from these fibrillæ. I would observe, in passing, that the vessels which remain do not present a trace of inflammation, there being simply, in certain points, a hyaline transformation of the capillary wall." Chaslin espouses certain views of Ranvier, and maintains that a distinction should be made between the connective tissue of mesodermic origin and the neuroglia, which is of epithelial or ectodermic origin; the latter embracing the Müller's fibres of the retina, the fibres and cells of the neuroglia in the spinal cord, and the slightly differentiated prolongations of the spider-cells in the brain. This peculiar neurogliar sclerosis, which he described for the first time, is, he claims, entirely distinct from a sclerosis of the connective or mesodermic tissue, as the peculiar fibrillæ distinctly emanate from the neuroglia-cells, whilst the non-adherence of the pia mater and the relative integrity of the vessels are indirect proof in the same direction. He has also given histo-chemical proof of these assertions. These fibrillæ in the bundles resist, in sections made after immersion in bichromate, the successive action of a 40 per cent. solution of potassa for ten minutes, as well as washing with water and concentrated acetic acid, whilst they remained colored red by the picrocarmine used after washing in water, and they can be preserved thus in formic glycerin. When the connective tissue, however, is treated in this manner it swells and decolorizes so that in a section of the spinal cord the pia mater will be swollen and decolorized whilst the neuroglia is intact. Moreover, after the action of alcohol diluted to one-third these fibrillæ remained colored by the carmine when subjected to acetic acid, whilst all other forms of the connective tissue of the body were decolorized. So definitely and so judiciously is this pathological distinction of Chaslin's given that it merits attention. The author believes that this neurogliar sclerosis is the same as that which has been found in the cornu ammonis and in the olivary bodies in the same cases of epilepsy; but if it is, certainly his localization of it in the first layer of the cortex gives it an importance and possibility that were never inherent to any former descriptions. Bevan Lewis has described certain striking degenerative changes observed in the cells of the second layer of the cortex, and in some cases this has been found to affect all the layers, even including the series of spider-cells. These are described in detail in the chapter upon the "Pathology of Insanity."

Our knowledge of the *modus operandi* of epilepsy is not much more definite than our knowledge of the exact pathology. The medulla oblongata was the portion of the nervous system that the older writers held to be most concerned in the production of symptoms. Van der Kolk believed that the nuclei of origin of the hypoglossal nerve in the floor of the fourth ventricle were always in a condition of induration which was proportionate to the amount of tongue-biting that had been observed in the attacks. But this was the airiest of theories, and is scarcely worthy of mention, much less of sober discussion. Then Nothnagel asserted that in the floor of the fourth ventricle there was a certain area which he called the con-

vulsive centre (*Krampfcentrum*), and that irritation of this caused epileptic convulsions. He regarded this area as the vasomotor centre of the medulla oblongata. The location of this area coincides, according to Wernicke, with the lateral motor field of the tegmentum in the pons, the lateral portion of *Frtg* in Figs. 42, 43, 44, and 45, its lower boundary being formed by the pons, its upper boundary not having been determined. A simple section will suffice as irritation, and the deeper the section the more are the lower extremities convulsed, and the nearer the section approaches to the corpora quadrigemina the more violent and generalized become the convulsions. Complete section of this region did not produce convulsions nor death by bleeding. Owsjannikow has demonstrated, however, that this area of Nothnagel's is the point of termination of the so-called long nerve-strands in which run the fibres of the upper and lower extremities, possibly concentrating at this point in a collection of ganglion-cells; so that Nothnagel's observation simply added another fact to our knowledge of the various focal lesions capable of causing epilepsy. During the latter years in which our knowledge of the functions of the cortex has received so tremendous an impulse from the experiments of Fritsch and Hitzig facts have accumulated tending to demonstrate more and more clearly that epilepsy in a large number of cases, if not in all, is due to direct or indirect excitation of the cortex or of the nerve-strands leading from the cortex to the peripheral structures, and Nothnagel's observation, viewed in the light of these later facts, only demonstrating the existence of a centre subsidiary to that of the motor cortex, but similar in kind. It has been shown again and again that electrical or mechanical irritation of the motor centres can cause convulsive seizures of tonic and clonic nature with loss of consciousness, and it has also been demonstrated that the same effects can be produced by the same irritation of the nerve-strands underlying the cortex. Duret has produced the most widespread convulsions also by irritating the cerebral membranes, more especially the dura mater. But we must remember, too, that epilepsy can be produced by such extracranial lesions as diseases of the spinal cord, of the peripheral nervous system, of many of the non-nervous viscera, by alterations in the blood such as are to be found in albuminuria and malnutrition, and by the action of many toxic agents and various febrile conditions; so that it would be a matter of large assumption to say that in all these various conditions the cortex of the brain alone is the part affected. The truth probably is that the epileptic manifestations are due to a peculiar molecular condition of the motor tract which runs from the motor convolutions to the peripheral motor structures and muscles. We are in entire ignorance of this exact molecular condition, as we are in entire ignorance of the exact molecular condition producing chorea, or neuralgia, or tetanus, or hysteria, or any one of the different functional nervous diseases, and we shall probably remain in such ignorance until in the ripeness of time we shall possess such instruments of precision as will enable us to see the molecular play in the living brain and spinal cord, or until we shall have so

advanced in our methods of preparation and staining of the different constituents of the nervous system that we can detect such slight cellular alterations as have thus far entirely eluded our vision. There are many childish metaphysical theories that presume to explain these cellular alterations, but they are simply laughable in the present condition of our knowledge. Whatever this altered molecular condition may be, there can be no question that it finds its expression in epilepsy through the motor tract running from the motor convolutions to the motor structures, the muscles of the periphery. When muscles are convulsed they can be convulsed only by direct excitation of the muscle itself, or of the motor tract leading from the muscle up to the motor convolutions, or of these convolutions themselves. But some varieties of epilepsy are evidently due to an excitation that extends into this motor tract from some part of the nervous system beyond it. Some cases, for example, present such symptoms as hemianopsia, word-deafness, aphasia, or the propulsiveness of propulsive epilepsy, indicating a lesion in the corresponding centres of the cortex or cerebellum or their underlying nerve-strands, and autopsies have demonstrated the correctness of this view; whilst the epileptic convulsions that are observed from lesions of non-nervous organs, diathetic conditions, and the action of toxic agents, must be from direct implication of this motor tract.

Idiopathic epilepsy, therefore, is a neurosis, like neuralgia, migraine, hysteria, and chorea, and it is exceedingly questionable whether the sclerotic and cellular changes that have been found are not effects rather than causes.

Diagnosis. The reader should fix in his mind firmly the fact that epilepsy is a symptom, and that therefore the burning question of diagnosis is to determine of what epilepsy is a symptom. We should always seek to ascertain, therefore, whether it is the symptom of any organic disease, such as lesions of the brain, spinal cord, or of the peripheral nerves; whether it accompanies the cerebral palsies of childhood; or whether it bears a distinct relationship to heart disease, marked gastro-intestinal disturbances, nephritis, or malnutrition.

When all these organic lesions have been excluded the epilepsy may be said to be idiopathic. Its diagnosis should not then be a matter of difficulty. The convulsions consist of unconsciousness, with tonic or clonic movements, or simply of loss or impairment of consciousness, either without convulsions or very slight convulsive muscular movements, and the differentiation need only be from hysteria and simulation.

True hysterical attacks have not the same sharp loss of consciousness that occurs in epilepsy, and hysterics do not, therefore, fall and bruise themselves or bite the tongue as do epileptics. Moreover, the movements of hysteria are more volitional and of wider range, so that the patient rolls and turns herself in bed or assumes various attitudes, whilst in epilepsy the muscular movements are limited in range, consisting only of limited flexions or extensions or simple rigidity of short duration with a slight tremor. It should not, how-

ever, be forgotten that true epilepsy may be conjoined with true hysteria in the so-called hystero-epilepsy.

The simulation of epilepsy is extremely rare in this country, except in certain prisons where it is known that the patient afflicted with this disease might be transferred to more desirable quarters in a hospital or an asylum for the criminal insane. In Europe it is said to be frequent among those desiring to escape military service. A number of attempts have been made by different authors to determine symptoms that are absolutely pathognomonic of epilepsy, but these have all been failures. Marc thought that holding some asafœtida under the nose would not increase the paroxysms of a true epileptic, but this has been disproved. Voisin has laid great stress upon the peculiar characteristics of the pulse during, before, and after the attacks of epilepsy, but these have been found to exist in other conditions, and Féré has demonstrated that they were absent in certain epileptics, and varied very little in others. Among the English thieves there is a class known as "dummy chuckers," whose business it is to simulate a convulsion in a crowded place and thus permit their confederates to rifle the pockets of the sympathetic bystanders. One of this undesirable class honored our American prisons with his presence for a number of years, and I have frequently had him "chuck a dummy"—as simulating an epileptic convulsion is called in the thieves' parlance—for my class; and I must confess that when this simulation is artistically done the fraud cannot be detected except by close and repeated observation, when the unchanged color of the face, the undilated pupils, the lack of the long sleep afterward, and the tendency to overdo the clonicity or the tonicity of the convulsions or the bleeding of the tongue (which is generally cut beforehand with a sharp knife), may throw light upon the matter.

PROGNOSIS. It is as yet undetermined what shall constitute a cure of epilepsy. In some cases entire immunity from attack may extend over a period of ten, fifteen, or even twenty years, whilst a convulsion in infancy may not be followed by any further attacks for many years, when a series of them may set in. Sometimes remissions of several years may occur, as I have known in my own practice, even as long as six or seven years. In other and less favorable cases the life of the patient may be made more pleasant and useful by a lessening of the frequency and violence of the attacks. In some cases, however, no improvement can be effected by any means now known to the medical profession. *Grand mal* may be improved in most cases, but in only a few of them can a cure be effected in the sense in which I have spoken of it. Experimentation by treatment of a month or so is the only way in which it can be ascertained what proportion of improvement can be effected. The relatives of the patient should always be informed that continuous treatment for years must be kept up to secure even an improvement. I have never yet seen a case of *petit mal* materially affected by treatment, except those that occur from malnutrition and the formation of ptomaïnes from an acute indigestion. The prognosis of the milder

degrees of hystero-epilepsy is usually excellent if proper treatment and control can be afforded the patient. Cases associated with migraine may also be greatly improved by treatment, and those of peripheral origin usually improve under treatment, although a convulsive tendency is generally apt to cling to the latter.

Treatment. The treatment of epilepsy will vary somewhat in the different varieties of the disease. Cases caused by removable intracranial lesions should be operated upon if the localization of the latter can be made by our present knowledge of the subject. The cortex of the cerebrum and the cerebellum, the centrum ovale of the cerebrum, and all of the subcortical tissue of the cerebellum may be easily reached by the surgeon, and our knowledge of cerebral localization will be a reliable guide to him. The nature of the lesion, however, should be determined before any operation is attempted. In the hemiplegias, single or double, and in the paraplegias constituting the so-called palsies of childhood it would be folly to operate, as they are due to the cerebral losses of substance known as porencephalitis, to hemorrhage, to arterial or venous thrombosis, or sclerosis and encephalitis, which usually proceeds from preceding arterial trouble. In cases of well-defined tumor an operation should always be performed. An operation is eminently practicable in cases of abscess, if it can be localized in an accessible area. In cases of epilepsy caused by cerebral trauma the scalp should be carefully shaved, careful search made for the cicatrix or depression of the skull, and, if either is detected, trephining should always be done, for in many cases slight adhesions of the membranes have been removed with excellent results. Trephining is a harmless procedure under proper antiseptic precautions, and may disclose conditions which otherwise could not have been brought to light. It is exceedingly questionable, however, whether the epileptic habit can be cured by an operation of any kind. Thus, Dr. E. G. Mason has collected for me 44 cases operated on by different surgeons, and the results were as follows:

Cured	2
No improvement	10
Death due to operation	3
Improvement, return of fits: malignant brain disease	1
Cases in which there was a cessation of fits one year after operation, but in which bromides were used	2
Temporary improvement; time of observation, however, not given	3
Cases in which time of observation after operation is sufficient to make them of value in statistical tables	23
Grand total	44

(This table can be found in Dercum's *System of Nervous Diseases*, Art. "Epilepsy".)

If peripheral irritation exists in cases of epilepsy, the irritant should be removed, even though the improvement obtained should be only temporary, as even this may be a valuable adjunct to the treatment by medication. An adherent prepuce or phimosis should be rectified. An irritable clitoris should be treated either by careful application of nitrate of silver (a 60 per cent. solution), or by soothing applications. If vaginitis exists, it should be carefully treated. Marked errors of ocular refraction should be remedied, but the

removal of the insufficiency of ocular muscles is, in my opinion, quite useless. If a laryngeal tumor exists, giving rise to the symptoms of so-called laryngeal vertigo or epilepsy, an operation should be resorted to for its removal. In all these cases surgical treatment alone should not be relied upon, as it is very difficult to eradicate the epileptic tendency when once established.

Parents are too apt to spoil and pet their children when afflicted with hysteria or epilepsy, and as a result the little patients come to have even less than the usual feeble self-control of childhood, and too often manifest almost savage tendencies. Careful and firm measures should be adopted in such cases, either by the aid of a trained nurse, or in a thousand ways that may suggest themselves to the good sense and tact of the physician or parents. Moral treatment in cases of this kind is often most surprising in its effect for good.

Migraine should be carefully treated if it exists in conjunction with epilepsy. Full directions for such treatment will be found in Chapter IX., under "Migraine."

If upon inquiry a tendency to periodicity is discovered, at such periods the patient should be kept quiet and special care paid to the medication. In many cases where this periodicity is manifested I put the patient to bed during the time of the expected attack, and either increase the quantity of the drug being administered, or add to the medication. In nocturnal cases a dose at bedtime may be all that is necessary. In some of these patients the attacks may be diverted from their usual period of recurrence, so that they may occur at some other time in the month, or an attack may come in the daytime. In such cases continuous treatment should be kept up, simply paying attention to the periods in the manner indicated.

The bromides, without doubt, constitute the most valuable means of treatment at our disposal. Unless the idiosyncrasy of the patient prohibits it, they should be given in large doses. In some epileptic patients the bromides are badly borne, but, as a rule, they will bear well all that it may be necessary to administer. In some exceptional cases the disease seems to be aggravated by the use of a bromide; in others an ounce may be given without ill effect; whilst in a few a collapse will be produced by a dose of 10 grains. Taking all these facts into consideration, my plan of treatment is as follows: The dose of the bromide of potassium is at first 10 grains three times a day, unless the attack is markedly periodic, in which cases I administer the bromide in proportionately large doses. My usual prescription is as follows:

℞.—Potass. brom. . . . . ℥ss.
Aquæ . . . . ℥iv. M.

Ft. sol. S.—Teaspoonful three times daily, after meals, in half-tumbler of water.

Unless improvement is manifest, or bromism has taken place, I increase the dose to 15 grains three times a day, after about a week of treatment by the smaller dose. I still further increase the dose to as much as 30 to 40 grains daily, in case the patient bears the bromide

well, but does not improve. If the manifestations of epilepsy do not lessen, but symptoms of bromism are seen, I conjoin with each dose of the bromide of potassium 5 grains of the bromide of sodium, as in the following prescription:

℞.—Potass. brom. . . . . ℥ss.
Sodæ brom. . . . . ʒij.
Aquæ . . . . ℥iv. M.
Ft. sol. S.—Teaspoonful three times daily, in half-tumbler of water.

This combination of the two bromides will often increase the effect upon the disease without increasing the constitutional effect of the drug. This dosage should be continued, if the patient has been satisfactorily brought under its control. If relapses occur, or improvement ceases, I try change of scene, a laxative, a brisk cathartic, or a combination with the bromide treatment, as already described, of borax, belladonna, or hyoscyamine. These latter means I use for a short time only, gradually discontinuing their use. These are embodied in the following prescriptions:

℞.—Potass. brom. }
Sodæ borat. } . . . . . āā ℥ss.
Aquæ . . . . ℥iv. M.
Ft. sol. S.—Teaspoonful three times daily, after meals, in half-tumbler of water.

Or,

℞.—Potass. brom. }
Sodæ borat. } . . . . . āā ℥ss.
Bellad. ext. fld. . . . . ʒss.
Aquæ . . . . ℥iv. M.
Ft. sol. S.—Teaspoonful three times daily, after meals, in half-tumbler of water.

The hyoscyamine should be given alone, in tablet triturate (gr. $\frac{1}{100}$ night and morning); Merck's preparation should be used. The loss of uvular reflex has been thought by some authors to be of great importance, and the ability to tickle the patient's throat with a feather without causing him to gag is taken as an indication that sufficient bromide has been administered. In my opinion, this is of little value, as I have known cases to grow worse when this reflex had been abolished, and I have seen improvement in others where the reflex was still present, and in still others the bromide has been well borne in increasing doses even though the reflex had ceased. The bromide of potassium and the bromide of sodium are the only ones of which I make use, as the bromide of ammonium has not proved of any use in my hands, except occasionally as an adjuvant. The acne, which is sometimes considerable, has been usually overcome by moderate doses of Fowler's solution of arsenic—gtt. ij–iij, three times daily, in a wineglass of water. It has been my experience that cases which are not benefited by the bromide treatment will fail to improve under any other, the only exceptions being some few cases that will do well with borax when the bromide has failed.

Gowers' maximum-dose treatment is occasionally very useful, especially in alternation with the treatment just detailed. This consists of doses of 2 or 3 drachms of bromide of potassium every second or third morning, increasing the dose to 4 drachms every fourth morning, and 6 drachms or an ounce every fifth morning, these doses being given after breakfast in a tumblerful of water, as, if they are not well diluted, epigastric pain and vomiting may be caused. He does not increase the dose beyond that which produces transient lethargy and dulness. There is great variability in the susceptibility of patients to the doses, some being unable to bear more than 4 drachms, whilst in others, as I have myself observed, no unpleasant symptoms will be produced by the administration of an ounce. The maximum dose should be reached in two or three weeks, and repeated three or four times, gradually reducing the doses so that six or seven weeks will be consumed in the treatment, when the patient may be left without treatment for several weeks or even months, although I have never seen such long periods of immunity as Dr. Gowers claims.

Belladonna, except as an adjuvant, has not proved of much use in my hands. The dose should be 1 or 2 minims of the fluid extract three times a day, carefully watching the effects of the drug. In some cases 10, 20, or 30 grains of borax thrice daily, well diluted, is of as much efficacy as the bromide treatment, though not generally so. The borax treatment (gr. 10 to 20, three times daily, in half-tumbler of water), however, should be carefully tried in all cases where the bromides fail in effect, or disagree with the patient.

In the last few years, however, the salts of strontium, the lactate and the bromide (gr. 10 to 20, three times daily, in half-tumbler of water), have been spoken of as having considerable value in the treatment of epilepsy, the dose being the same as that of the bromides. I have not, however, had any extended experience with them, and can, therefore, only mention the fact. Nitrite of amyl is sometimes useful in arresting an attack which has been preceded by an aura or warning of sufficient length to permit it to be used, but this seldom occurs; indeed, I have almost abandoned the use of the drug. The zinc preparations, which were so much in vogue twenty years ago, have been of very little value in my cases.

MIGRAINE.

Synonyms: Hemicrania. Sick headache.

Migraine is a very frequent affection. In the typical form the symptoms, as the name indicates, affect one side of the head; but there are many variations from the type, and the most frequent is that the pain commences in a certain spot, most frequently over the eyebrows or the vertex. Usually the onset is gradual. The patient wakes in the morning with a certain feeling of lassitude and uneasiness about the head that experience has shown to be prodromal. As the day goes on, this feeling merges from discomfort into actual pain, and this develops until the afternoon or evening,

then reaching its acme, when the pain becomes severe, at times almost unbearable, and culminates in one or several attacks of vomiting, after which it gradually diminishes to terminate suddenly or gradually. The face may either be flushed or pale, the pupils dilated or contracted. In the non-typical forms, however, the pain may vary very greatly in severity and duration. Thus, in some there may be simply a feeling of uneasiness which may last a whole day or several days, or only a few hours. In some, too, there is no vomiting, and its place may be taken by nausea, or even this may be lacking. The attacks usually return with a certain irregular periodicity, most frequently within the month. In women they are especially prone to bear some relation to the menstrual period, coming either before, during, or after it, and this does not necessarily denote any uterine trouble. Migraine bears a curious relationship to epilepsy. In almost every case that has been subject to migraine throughout many years there will be found to have been some loss of consciousness at some time, which may have only been called a fainting-fit. In others, however, there may occasionally be a true loss of consciousness, conjoined with muscular convulsions of the true epileptic type. In still others the migraine may regularly alternate with epilepsy, so that when one is present the other is absent, and *vice versa.* This latter form is comparatively rare. The attacks generally first appear in adolescence or early adult life, and usually continue until late in life, often gradually passing away as old age approaches. Individuals who are subject to migraine are often subject to alternations of curious functional disturbances. Thus, one patient may have, in place of the migraine or after it, a continuous depression of spirits that has none of the other symptoms of melancholia, such as insomnia, post-cervical ache, or suicidal tendency, or the *facies;* or another may have a persistent tachycardia, or another may be possessed of a persistent general restlessness and nervousness or irritability. In some patients subject to migraine life is passed in a constant round of migraine and its alternating nervous conditions. Patients who are subject to migraine have no mental impairment; indeed, I think that most of the patients whom I have observed were above the normal in their mental capacity. I could name from memory a formidable list of distinguished public men—lawyers, physicians, artists, inventors, authors, and others of high intellectual stature—who are afflicted with migraine, and in whom the disease in no wise impairs the mental capacity except that at the time of severe manifestations the patient is sometimes temporarily confused, and even in some extreme cases slightly dazed. A number of years ago I was concerned in the trial of a very important will case, and one of the leading counsel was to my knowledge subject to violent attacks of this malady. One morning upon going to court, when the legal gentleman whom I have mentioned was to commence an important examination of me as a witness, I immediately perceived that he was very pale, that his pupils were dilated, and that he was slightly dazed. I at once recognized the fact that he must have had a severe attack of migraine the night before, and that he was in no condition

to enter upon the intellectual sword-play necessary for the day's work, and I called one of the other counsel to me, stated the fact, and upon another plea had the court adjourned for the day, after which I found out that my friend had been up all night, as I had supposed, with a violent attack, and that he was scarcely conscious of what he was doing. The next day, however, he took his place, and went as brilliantly through the fray as could have been expected. I cite this as a striking example of what is occasionally observed. In certain cases of migraine there is impairment of vision, which may consist of slight dimness or irregular blurring, or even of hemiopia, and these may sometimes persist for several days after the attack, but that they are functional may readily be determined by the history of the case.

The chief cause of migraine is heredity, and I have discovered a singular fact, namely, that the heredity is mainly through the mother. It may, however, come through the father, although this is rare, and much more so than to see it descend through both father and mother. I have never seen a case that was not hereditary—*i. e.*, in which there was not either a heredity of migraine or epilepsy, or some neurosis. The exciting cause may be fatigue, the emotions, and any cause that may depress the general health.

The pathology of migraine is unknown. It is in my estimation a neurosis—that is to say, a temporary disease that is not accompanied by gross lesions. It stands midway between a neuralgia and an epilepsy, and it may be called a neurotic neuralgia or a neuralgic neurosis. There has been, for many years, a question as to whether it was of vasomotor origin, and an attempt was made to base this upon the coloration of the face; but as this hue varies very greatly in different cases, and as there has been no proof whatsoever of the vasomotor origin, this theory remains a pure theory, worthless for any practical purposes.

The prognosis of migraine is excellent so far as regards life or a tendency to pass into other nervous affections, for I have never known a case to die from it, or to pass into any other nervous disease, except in the very exceptional cases of alternation with epilepsy. In this conjunction it must be remembered that the occasional losses of consciousness of which I have spoken are of no consequence, and should not be for a moment regarded as true epilepsy. The prognosis as regards the cure, however, is extremely doubtful, for whilst these cases can often be greatly benefited, I have never known of one being cured. They occasionally die out, however, in middle age.

The diagnosis of migraine is very easy, because it is an affection that recurs with an irregular periodicity, with a gradual or sudden onset, consisting merely of a gradually developing paroxysm of pain around the head, generally unilateral at the outset, that culminates in severe or agonizing pain which may be accompanied by nausea or vomiting. In certain cases, however, where the health has been very much depressed, it may recur so freqnently and so violently as to lead to a suspicion of an organic trouble, so that there may be a question of brain tumor; but this can readily be excluded by the

irregular periodicity of the affection, by the isolated attacks, by the lack of retinal changes, by the lack of any paralytic symptoms, and by the absence of any mental impairment.

The treatment of migraine should be directed to the amelioration of the disease itself, as well as to lessening the suffering of the individual attacks. The individual attacks cannot be cut short by remedies administered at the time. This may be laid down as an axiom. They can, however, often be mitigated considerably. The best of all drugs, in my opinion, is antipyrin, and five or ten grains of it administered every hour or two until the pain lessens will generally act like a charm. This should be administered in water or in the form of tablet triturate or compressed tablet. The objection to antipyrin, however, is that it is very apt to depress, and inasmuch as this is a recurring disease, the habit of taking antipyrin is a dangerous one. Next to antipyrin, bromide of potash in doses of ten, twenty, or thirty grains at the outset of the attack is the best drug, and this should be administered in solution, being given in at least a tumblerful of water. It is not nearly so depressing as the antipyrin, and the danger of taking it in recurring attacks is usually very small, except in unusually asthenic individuals. It should be determined what dose will give relief, and this should be administered promptly as soon as the attack has commenced, best of all early in the morning, when the first prodromal symptoms are observed, and a second dose may possibly need to be given during the day. If the pain is agonizing, antipyrin may be given in conjunction with it; but, as I have said, this should be done with caution, and the drug should not be placed in the patient's hands for indiscriminate use. In some few cases, unfortunately exceptional, large and repeated doses of a slightly alkaline mixture will cut short an attack, and this may be done by the spirit of Mindererus, half an ounce every hour or two in a wineglass of water, or a glass of Vichy with a pinch of baking-soda in it every hour or two. In my experience, however, these alkaline mixtures may act well in one or two attacks, but seldom in more than this. They are worth trying, however, at times. Guarana (ext. guaranæ fluid., gtt. x to xx, in water) is occasionally an excellent remedy, but it is not nearly so reliable in my experience as bromide or antipyrin. Peppermint, applied externally, often gives great relief for a time. Use should be made of the oil of peppermint on a piece of absorbent cotton, care being taken that it does not blister the skin; or the menthol pencils are very convenient, brushed lightly over the skin. Phenacetine, in tablet-triturate form, is often a famous drug for giving transient relief, and the dose should be five grains every hour until relief is given, or fifteen grains have been administered. It does not depress much, as a rule, but it is not so reliable as antipyrin. Occasionally a full dose of Huxham's tincture, ʒss–j, will abort an attack. Hare recommends a pill containing acetanilide, gr. ij, camphor monobromate, gr. $\frac{1}{2}$, and citrate of caffeine, gr. $\frac{1}{2}$. It may be well to try one after another of these remedies in a difficult case, as the reaction of patients is extremely variable. In some very severe and agonizing

cases resort must be had to a hypodermic of the sulphate of morphine (gr. $\frac{1}{8}$ to $\frac{1}{4}$); but this needs careful consideration, and should never be administered except by the physician. If the pain recurs in such violent paroxysms as to prostrate the patient, causing coldness of the extremities and rapid pulse-beat, and this cannot be controlled by antipyrin and bromide, hypodermics of morphine should be unhesitatingly administered, for unless the habit of the pain is thoroughly broken up it cannot be controlled afterward. For this reason it will usually not be necessary to administer the hypodermic in more than a few attacks until such time as the pain has again been brought within bearable limits and recurs without other grave nervous disturbances, after which resort may again be had to bromide, antipyrin, guarana, the salines, etc. During these severe attacks the patient should be placed in bed, and warm applications—best, of hot bottles—should be applied to the hands and extremities. Not only is this application of heat very grateful to the patient, but it lessens the pain. In every severe case the action of these special remedies may be aided by keeping the patient in bed, quiet, with the room darkened, although this extreme measure may not be necessary in the mild attacks. In some slight cases the attack may be cut short if, at the time of the first prodromal symptoms, a dose of bromide, 20 to 30 grains, is taken, and the patient then lies down in bed for a few hours, with or without sleep, the room being darkened and noises being carefully excluded. In the treatment of the disease itself other remedies are needed. The best one, in my opinion, is cannabis indica, and this is best administered in the form of the extract, of which $\frac{1}{4}$ to $\frac{1}{3}$ grain should be given three times a day continuously for weeks or months, as in the following prescription :

℞.—Ext. cannab. ind. , ... gr. vj.
Ft. in pil. no. xxiv. S.—One three times daily.

I believe that cannabis indica acts as well in migraine as the bromide does in epilepsy, and it should be as continuously used. With the cannabis indica it may often be found useful to administer a dose once a day, generally at night after the evening meal ; of the bromide of potash, 10 to 15 grains, although in many cases the extract of cannabis indica alone will suffice. Although the bromide is recommended as being of value in the treatment of migraine when used continuously, I cannot say that this has been my experience, because I have found that it is not nearly so reliable as the cannabis indica ; moreover, I am decidedly opposed to continuous doses of the bromides in any case wherein it is undesirable to impair in the slightest degree the patient's intellectual faculties, for I do not believe that any living human being can continuously take the bromides without having a subtle loss of the highest intellectual capacity—losing, as it were, the fine blush of the mind. I have spoken of this more particularly in the section upon "Epilepsy." Indeed, so much value do I attach to the extract of cannabis indica, that if relief is not obtained in a severe case of migraine by its use, I should advise that

resort be had to other measures than the bromides. In the first place, the general health should be carefully attended to. If there is any anæmia, this should be overcome by the use of iron in full doses, and it must always be remembered that the dose of iron varies very greatly in the different ferruginous preparations. The dialysed iron, either the ordinary dialysed form or Dree's albuminate of iron, should be administered in drachm doses three times a day after meals, and this should be administered in a *cup* of water, and not in a tumbler, because the dark hue of the iron is in some fastidious individuals apt to produce a sensation of nausea if seen in a transparent vessel like a tumbler, whilst it can be pleasantly taken in a cup. This is a matter of small clinical detail that often makes a great difference. The peptonate of iron and ferratin are the best preparations, however; the former in doses of 3 grains three times daily, in the form of compressed tablet, and of the latter gr. iv–viij, in capsules. If the anæmia is pronounced, the citrate of iron and quinine, 3 to 5 grains three times a day in pill or tablet-triturate form, is an excellent remedy, although in some cases in which the anæmia has very greatly depressed the general health it will be advisable to administer one of the preparations of iron that I have mentioned in conjunction with 2 or 3 grains of the sulphate of quinine in tablet triturate, pills, or capsules. In some cases of anæmia in which there have been marked hepatic symptoms, or in which there is a yellowness of the skin and conjunctiva, the iron will have a much better effect if it is not only conjoined with quinine, but also with the mercury, and a perfectly reliable preparation of the latter is the bichloride, of which $\frac{1}{60}$ to $\frac{1}{32}$ grain should be taken three times a day, either in solution, tablet triturates, or pills. This is another small clinical detail that is of considerable value in my opinion, and its therapeutic importance is probably due to the fact that in increasing the hepatic secretions it also increases the blood-corpuscles in some subtle way. I am perfectly well aware that mercury is said to decrease the number of the red corpuscles, and yet I have again and again observed the clinical fact, of which I can give no explanation. In addition to these medicinal measures for the relief of anæmia the patient should take large quantities of nitrogenized food, eating savorily prepared hot meat at least three times a day, and in extreme cases using the equivalent of a pound of beef made into beef-tea or a beef-extract; sometimes, also, bullock's blood that can be obtained from any slaughter-house, and that can be frozen into chunks, which are by no means unpleasant to eat after the patient has overcome the prejudice. Besides this, the expenditure of energy should be cut off by restricting the amount of exercise, and keeping the patient in bed until late in the morning, or even until noonday. This latter measure, however, will not be necessary except in extreme cases. If there is lithæmia, the treatment should be something entirely different; but lithæmia should not be diagnosed from the presence of a small amount of uric acid, or from a few oxalate of lime crystals in the urine, but rather from the fact that the patient has been known to be taking entirely too much food or has passed gall-stones, or

because the urine is constantly loaded with large amounts of uric acid and the amorphous urates. I have seen many cases of anæmia mistaken for lithæmia. In every doubtful case it is an excellent rule to apply a stethoscope to the heart and the large vessels coming off from the heart, and I have more than once seen the attending physician by whom I have been called in consultation perfectly amazed at detecting loud blowing anæmic murmurs in a case that had been treated for lithæmia. If there is true lithæmia, however, the treatment should be begun by a moderate dose of calomel, say 3 grains, with 15 grains of the bicarbonate of soda, given at bedtime, and if this has not acted the next morning before breakfast, a slight saline laxative should be given, such as a wineglassful or half-tumblerful of Hunyadi, or 2 drachms to $\frac{1}{2}$ ounce of the sulphate of magnesia. Either an acid or an alkali should be administered for a week or ten days. The best acid, I think, is the dilute nitro-muriatic, of which the dose should be 20 drops three times a day after meals in a wineglass of water, as follows:

℞.—Acid. nitro-muriat. dil.	℥j.	
Aquæ	℥ij.	M.

Ft. sol. S.—Teaspoonful after meals in a wineglass of water.

In some cases, however, an alkali will do better than an acid, and I know of no means of determining this except by that of actual experimentation. The best alkali is a glass of Vichy or Giesshübler, with a pinch of baking-soda in it, three times a day, an hour or two after meals. The bowels should be kept open, but not more than one natural movement in the day should be maintained, and it is sometimes a considerable study as to how this should be done. A glass of Hunyadi may answer, or it may be necessary to administer the cascara sagrada, 2 grains of the solid extract in pill-form, or a drachm of the fluid extract. This remedy varies very greatly in its effect in different persons, and it should be determined by observation what the necessary dose should be, for it may be necessary to give 2 grains of the solid extract or a drachm of the fluid extract three times a day, or even to treble or quadruple the dose, and the great advantage of the cascara sagrada is that this increase of the dose when necessary is not accompanied by any griping or other untoward symptoms. In some cases the best laxative is this:

℞.—Ext. aloës, Ext. hyoscyami . . . . . āā	℈j.	
Ext. nucis vomicæ	gr. v.	M.

Ft. in pil (argent) no. xv. S.—One daily after dinner.

If in lithæmia the alkalies or the acid cause slight depression, as they are very apt to do, $\frac{1}{60}$ to $\frac{1}{30}$ grain sulphate of strychnine should be administered with them, and a favorite formula of mine is:

℞.—Acid. nitro-muriat. dil.	℥j.	
Strychniæ sulph.	gr. $\frac{1}{2}$.	
Aquæ	℥ij.	M.

Ft. sol. S.—Teaspoonful after meals in a wineglass of water.

For further details of more pronounced cases of lithæmia the reader should turn to the section on "Neurasthenia." If, in spite of this attention to the general health, the attacks keep recurring, great relief may often be obtained by overcoming any error of refraction. In some cases where there is no great error of refraction, but in which there is exophoria, the use of prisms or even cutting the muscles of the eye is often followed by great relief. Although I have never seen a case of migraine cured by such means, I have yet seen many cases in which recurring serious attacks were broken up and the disease made to travel along in a very bearable manner. Travel and change of scene are often of great benefit in cases of migraine that have become severe.

CHOREA.

Synonyms: St. Vitus's dance. St. Anthony's dance. St. Guy's dance.

Definition. Chorea is a functional nervous disease characterized by involuntary or mainly involuntary fibrillary movements consisting either of a jerky twitch that begins quickly and ends quickly, or of a wavy, undulating movement.

History. Various functional phenomena of the nervous system have been called chorea, although they have borne very slight resemblance to what the neurologists of the present day call by this name. Thus the real dance of St. Guy or St. Vitus, which has also gone under the names of the dance of St. Modesti or St. John, choremania, orchestromania, chorea magna, chorea Germanorum, epilepsia saltatoria, or dance plague (*Tanzplage; Fléau de la danse*), was an epidemic prevalent at various times since the end of the fourteenth century, the first appearance of it having been described in 1374, although Hecker claims that it was no new disease, as it was well known in the Middle Ages. Men and women would form circles hand-in-hand and dance for hours together until they swooned or a state of exhaustion ensued, very much as is the custom in the ghost or Messiah dance of our North American Indians. They would then suffer greatly from tympanites, which was generally quickly relieved by abdominal compression with clothing or by thumping and trampling upon the abdomen. Vivid hallucinations generally accompanied these attacks, usually of a religious character, so that the heavens would seem to open and they would see the Saviour enthroned with the Virgin Mary. The attack would often commence with sudden loss of consciousness and the patient would fall to the ground, but would usually spring up to begin the dance with strange contortions. In 1374 this epidemic spread from Aix-la-Chapelle all over the neighboring Netherlands, and thence to Cologne and Metz. The communities were absolutely disorganized. In Cologne the number of the dancers amounted to five hundred, and in Metz to eleven hundred, all classes of the community taking part in the revels—peasants from their plows, mechanics from their workshops, housewives from their homes, girls and boys leaving their

parents, servants their masters, beggars and idle vagabonds seeking their profit in the general disorder; and it was four months before the Rhenish cities were able to gain control of the disorder accompanying the epidemic, although even then this went creeping on. This was called St. John's dance. When it broke out again in 1418 in Strasbourg it was denominated St. Vitus's dance, because the legend ran that St. Vitus, just before he suffered martyrdom with other early Christians in the time of Diocletian, had prayed to God that he would protect from the dancing mania all those who should solemnize the day of commemoration and fast upon its eve, and that a voice issued out of heaven saying that his prayer was accepted; so that St. Vitus became the patron saint of those afflicted with the dancing plague, as St. Martin, of Tours, was at one time of those suffering of smallpox, St. Anthony of those suffering with the "hellish fire," and St. Margaret of puerperal women. Paracelsus, in the beginning of the sixteenth century, was the first to make an intelligent examination of this disorder from a medical standpoint, and to recommend treatment, which consisted of harsh means, strict fasting, severe chastisements, immersions in cold water, and various nauseous compounds of the wonderful quintessences of that day. The disease appears then to have declined, and very little of it seems to have been known in the sixteenth century. The great English physician, Sydenham, gave the first accurate description of what we nowadays call chorea, usually giving it also his name. So true to the life, indeed, was his portrayal of the disease that nobody has ever surpassed it, and subsequent observation has merely amplified our knowledge of it.

Clinical History. Chorea consists of the following varieties:

- Sydenham's chorea;
- Athetoid chorea;
- Violent chorea;
- Chorea of pregnancy;
- Paretic chorea;
- Post-hemiplegic chorea;
- Post-hemiplegic and post-epileptic chorea;
- Chorea with loss of consciousness;
- Chorea with rapid pulse and rapid respiration;
- Chronic chorea.

Chorea is manifested by certain characteristic fibrillary movements which are varied somewhat in the Sydenham and in the athetoid form. In the former they consist of jerky, twitching movements, the jerk beginning quickly and ending quickly, whilst in the latter the action of the muscular fibrillæ is less abrupt and jerky, and is more wavy, approaching in miniature the gradual movement which has been observed in cases of athetosis, whence the name *athetoid*. In both forms the movements are only partially under the control of the will, to the extent that the patient can make them stop for a short time, and in some cases they cease during sleep. The fibrillary character of the movements is their salient characteristic, for in no other disease do we see anything exactly like it; and it is for this

reason also that simulation is possible, because the individual fibrillæ of muscle are not under voluntary control, although the body of the muscle itself may be. These fibrillary movements can be best observed by the examiner taking the patient's hands in his own, allowing them to rest palm downward lightly on his upturned palm, or removing the shoe and stocking, placing the foot in his lap, and observing the toes; or by stripping the patient and carefully watching the muscles of the trunk. The face does not usually constitute a good place for examination in the obscure cases. Where the choreic movements are slight, much patience must be exercised in these examinations, and I have held the hand of a patient for ten minutes before being rewarded by the characteristic twitch. The distribution of the chorea may be throughout all the muscles, although there is a tendency to one side being more affected than the other. The initial attacks generally occur in children at the age of nine, and in 250 cases of my own the ages ranged from two to twenty-eight.

The proportion of the sexes was as follows: females, 160; males, 90.

In violent chorea the movements are either of the ordinary Sydenham or athetoid form, and become violent, or they are violent from the onset. In this variety the movements preserve the jerky or the athetoid character—generally the former—but are far beyond the fibrillary range, affecting the whole body of the muscle, though in an automatic and involuntary way that is beyond all effort of simulation. So violent at times are the movements that the patient must be strapped in bed, or else someone must be placed on either side of it to prevent his being thrown out.

In certain cases of chorea of the Sydenham type there are losses of consciousness similar to those of the *petit mal* of epilepsy, without any convulsive movements, however, so far as I have seen. I have never seen this variety, with loss of consciousness, alluded to by any writer, and believe myself to be the first to describe it. It may be important or unimportant, according to the tendency of the chorea with which it is associated. If the chorea has the tendency to develop, of which I shall speak in a few minutes, the losses of consciousness may become very frequent, prolonged, and accompanied by convulsive movements of tonic or clonic character, and the patient may die in convulsions, with the symptoms of an acute encephalitis or meningitis. I have seen this in two cases, but only two of this type which have had a marked developmental tendency. If, however, the chorea shows no tendency to develop, or but a slight tendency, the losses of consciousness are harmless, and will invariably pass off under no further treatment than that addressed to the chorea. This has been my experience in six cases.

There is another peculiar form of chorea to which I have never seen any allusion made by writers upon this subject, namely, chorea suddenly developing after a slight attack of articular rheumatism, and associated with rapid pulse and rapid respiration. The history of the three cases which I have seen has been this: A slight

articular rheumatism had appeared, lasting only a few hours, and so evanescent that it might well have escaped the observation of a careless diagnostician. In the course of twenty-four hours a slight choreic movement began, and the respiration and pulse grew to be rapid, the former running up to 30 or 40, the latter to 120 or 130 or 140. Careful auscultation revealed no organic lesion of the heart or lungs. The chorea showed a marked tendency to develop rapidly, so that the movements increased greatly in intensity from day to day, and at the same time the tendency to develop manifested itself in the heart- and lung-beats, so that in a few days the choreic movements and the pulse and respiration made a dangerous triad of symptoms, the choreic movements, however, not developing in anything like the proportion of the increase in the pulse and respiration. In one of my cases the chorea ceased, but the pulse and respiration increased in rapidity, and the child died in the course of a week, a pericarditis appearing in the last forty-eight hours. In the other two cases the chorea never became very violent—never so marked, for instance, as in many cases of violent chorea, even at the onset, and yet the children died, neither of them developing endocarditis or pericarditis. No autopsy was permitted in either of the three cases.

The chorea of pregnancy is a rare disease, and I have never seen a case of it. It is said, however, to be very fatal, the percentage running as high as 33 per cent.

Chronic chorea is nothing more, in my estimation, than Sydenham or athetoid chorea or a violent chorea that has not been properly treated, so that the originally slight fibrillary movements have had superadded to them muscular movements of greater length, often with local spasms and contractures.

The course of a chorea is very inadequately understood by most physicians. Its duration is usually six weeks, this being the average computed in 250 cases of my own. But choreic movements invariably continue for months or years after a so-called cure, and the question of what is a cure is to a large extent an arbitrary matter. If patients are carefully examined as I have directed, it will be found that slight movements can be detected long after the parents suppose that a cure has been effected, and the continuance of these slight movements is the cause of so many of the odd grimaces which we see in adults as remnants of a chorea in childhood. Every year there is a tendency to relapse, for Weir Mitchell and Sinkler have shown that the greatest number of cases of chorea occurred in those months in which there are the largest number of cloudy days, the greatest amount of snowfall, and the greatest amount of barometric disturbance, whilst high or low barometer seems to have no effect upon the disease. In addition, they have shown that the proximity to the centre of the greatest barometric disturbance of a storm has a direct causative influence upon chorea. I can confirm these observations of Sinkler and Mitchell. It is well known, for instance, that a cyclone travels in an ellipse, being preceded by cloudy sky, foggy weather, and some slight barometric disturbance, whilst it is succeeded by what is known technically as the *anti-cyclone*, which is a period

of blue skies, light clouds, bright sunshine, and little or no wind. Now, I have found that some of these choreic patients are most disturbed by the approach of the storm, whilst others are most disturbed by the passage of the storm itself, and still others are affected the most during the period of anti-cyclone, or relapses may occur at these times.

Another important feature of the natural history of chorea is that certain cases have a tendency to develop, 25 cases among 250 displaying this characteristic. The cases which develop begin usually with pronounced symptoms and rapidly develop in a few days into violent chorea; but not infrequently the onset may be by slight symptoms of the minor type, and in a few days or a couple of weeks the development may be into a violent form. This developmental tendency should always be borne in mind, because much blame may attach to a physician if he has not forewarned the parents that this is one of the possibilities; and, moreover, a case which seems harmless at the beginning may become transformed into one of very serious import. And especially should the tendency to develop be cautiously considered in the cases beginning with slight losses of consciousness and in those accompanied by a rapid pulse and respiration.

CAUSATION. The causes of chorea are—

The seasons;
The emotions;
Trauma;
Articular rheumatism;
Malaria;
Imitation;
Heredity;
Eye-strain;
Race.

The effect of the seasons and barometric disturbance has been fully alluded to in the clinical history.

It is a very common thing to find that the emotions play a great *rôle* in the causation of chorea, and of all the emotions, fright is the most potent.

Trauma is one of the less frequent causes of chorea, but yet cases indubitably proceeding from this have been observed by me.

The *rôle* of articular rheumatism in the causation of chorea has been very much overestimated, and the idea that was formerly so prevalent that chorea was a rheumatic disease is entirely untenable. Thus articular rheumatism was a cause in only 18 cases out of 250 of my own observation, whilst 16 had functional cardiac disturbance and 22 valvular disease.

I have seen several cases of chorea with the typical malarial fever, and evidently caused by it, and these have usually been of short duration, ceasing upon the removal of the malaria.

Imitation has often been observed as a cause of chorea, especially by Mitchell.

Heredity is often of importance in chorea as an etiological factor. In a circular letter that was sent out by Dr. Mitchell a number of

years ago to physicians in the Southern States, it was shown that chorea was a very infrequent malady in the negro race. Whether this applies also to other races, I do not know.

Pathology. Although certain cerebral changes have been well described in the chronic and fatal cases, as well as of the disease in animals, there has never been any approach to a description of the pathological alterations that would explain the movements of the ordinary cases of chorea of the Sydenham and athetoid types. Hyaline degeneration in the nerve-cells of the central ganglia has been found by Meynert and Elischer. The capillary embolism produced by Angel Money by injections into the carotids of animals has not been verified in other cases. Eisenlohr in one case of congenital chorea found a sclerotic mass at the level of the third cervical nerve, and alludes to a similar pathological find of Schultz's. The best description of a fatal case of chorea is that given by Dana of a cerebrum, medulla, and spinal cord from a male patient of eighteen who had had choreic movements of the Sydenham type for over twelve years. There were chronic lepto-meningitis of the cerebrum, diffuse and varicose dilatations of the small arteries, especially of the deeper subcortical capsule; degenerative changes in the arterial wall, but no arteritis; the white matter was honeycombed with greatly dilated vascular lymph-spaces, and the cortical walls in most regions were normal. The severest changes, vascular, interstitial, and degenerative, were in the under surface of the temporal lobes, in the internal capsule and the adjacent parts of the corpus striatum (especially the lenticular nucleus), and the optic thalamus, and in this region varicose nerve-fibres were observed. In the pons Varolii and in the medulla a much less marked phase of the same conditions was found, and there was also a certain amount of cellular degeneration in some of the cranial-nerve nuclei and a slight increase in the connective tissue of the pyramids. In the spinal cord the pia mater and the cord were slightly congested, the latter especially in the lateral tracts, and there was a double central canal. In Dana's valuable paper upon this subject 200 autopsies are collected, made upon patients dying with acute or chronic chorea, but in only 39 of these was there a satisfactory account of the condition of the nervous system. From this it would appear that after death from chorea various lesions have been found in the nervous centres, such as congestions, extravasations, embolism, and softening, and that heart-lesions are very uniform. Of these 39 cases there were 25 of Sydenham chorea, uncomplicated with insanity or other serious disease. In 16 there are said to have been intense cerebral hyperæmia, peri-arterial exudations and erosions, softened spots, minute hemorrhages, and occasionally embolism, the changes being most marked in the lenticular nucleus and inner parts of the thalamus. In only a few cases was the cord affected somewhat similarly. In one case there was absolutely no change found, although Dana thinks some lesion was overlooked or a mistake made in some way. In one case only the corpus striatum and cord were examined, and in them there were foci of softening; and in three cases the brain was pultaceous, the lymph-walls were disintegrated and full

of micro-organisms. In six cases minute hyaline bodies were found, chiefly in the lenticular nucleus. In two cases of subacute chorea with insanity there was great cerebral congestion with thrombosis and minute hemorrhages. In five cases of chronic chorea of the Sydenham type lasting from four to twelve years there were found dilatation and thickening of arterioles, with necrotic and erosive changes, degenerative changes in nerve-cells, varicosities of nerve-fibres, old spots of sclerosis, and hyaline bodies, and a somewhat less implication of the spinal cord. In the dog a number of cases of chorea have been reported after section of the spinal cord, and some similar cases have been reported by Weir Mitchell in the human being. In 1875 Gowers examined the spinal cord of a choreic dog, and could find no alterations; but two years afterward, in conjunction with Sankey, he demonstrated that certain limited areas of the cord were infiltrated with small lymphoid cells. Putnam, however, could find no such alterations in two kittens which had been artificially made choreic. In one of these no changes whatever could be found in the nervous centres, but in the second there was a marked injection of the blood-vessels, and the ganglion-cells of the spinal cord and brain would not take up the staining-fluid, and could not be examined satisfactorily even in glycerin. H. C. Wood has examined the spinal cords of four dogs, three being killed while still able to go about. Two of them suffered from typical rhythmic chorea, one from chorea with movements exactly simulating those of a child, associated with great weakness. The fourth animal died of the disease after having completely lost control of his hind legs. In each of these there were numerous lymphoid cells in the gray matter, but very few, if any, of them in the white matter. In only one dog was any connection traced between these cells and the bloodvessels, and in this one instance there were some cells in the perivascular spaces, but there was no such outpouring of leucocytes as was found by Gowers and Sankey. Wood maintains, however, that these lymphoid cells are found in the normal cord. In Wood's dogs the ganglionic cells in the cord were profoundly affected, and he states that the inability of the ganglion-cells to take up the staining-fluid is present only in the earliest alterations in the cell. The cellular changes were greatest in the groups nearest the spinal canal and just posterior to it, whilst the lateral groups of very large cells which are seen in the dog's spinal cord were not so profoundly affected. In the first-named group the cells had been transformed into irregular, globous-looking masses, without sharp outline, and imbibing the staining-fluid very slightly. No granulations, no nuclei, no processes were present. These masses evidently represented the cells in the last stages of degeneration, and were of various sizes. The cells in the lateral group were distinctly altered. They took the staining very faintly; in a large proportion of them no nuclei could be found, and in many about one-fourth of their contents were occupied by a large mass of definite outline, free from granular contents, seemingly a vacuole. Of all the absurd theories that have been passed around from text-book to text-book and by word of mouth, the most absurd is that proposed by Broad-

bent and Tuckwell, of minute embolism of the cerebral arteries as explanatory of chorea. In the first place, these minute emboli of the cerebral vessels are extremely rare; in the second place, as Octavius Sturgis has succinctly pointed out, none of the symptoms of cerebral embolism are present in chorea, such as the sudden vertigo or headache, pain with faintness or sickness, hemiplegia, mental impairment, anæsthesia or aphasia, or a fatal termination by way of sudden coma. An attempt has been made of late years to show that chorea was due to some infection, and Richter has found cocci in the blood in one case, Donkin rod-like bodies in another, and Berkeley has thought that the pathological findings in another case were similar to those of diphtheria; but proof of this view is yet entirely lacking.

Differential Diagnosis. The characteristic twitch of Sydenham's chorea and athetoid chorea—the quickly beginning and quickly ending fibrillary jerk or the fibrillary wavy movement—is never seen in any other disease than chorea, whilst the muscular movements of larger range of violent chorea also have this quickly beginning and quickly ending or twitchy character. In chronic chorea, however, the movements may lose these characteristics and become intermingled with anomalous movements and with muscular spasms or contractures, but here a history of the antecedent chorea will invariably make clear the diagnosis. In all examinations, however, of patients who are suspected of chorea, not only should the toes and fingers be carefully and patiently examined, but the patient should be stripped so that the muscles of the trunk may be brought into play. Especially should this examination be carefully made in cases where there are slight losses of consciousness, or where there is a grunting or subdued sound that is present when the laryngeal muscles are affected, constituting the so-called laryngeal chorea, which is really an ordinary chorea, however, with implication of the laryngeal muscles.

Chorea should be differentiated from—

Organic cerebral disease;
Epilepsy;
Disseminated sclerosis;
The fibrillary tumor of progressive muscular atrophy;
Hysteria.

The differentiation from organic cerebral disease may become at first sight a matter of difficulty in cases where paresis is an early symptom or where there are slight losses of consciousness, but the detection of the fibrillary movements is all that is needed to make the diagnosis. Sometimes, too, the difficulty of speech, which is not infrequent in choreic children, may cause a suspicion of organic cerebral disease, but the movements make the matter plain.

The cases of chorea which are accompanied by slight losses of consciousness are rare, and therefore generally cause great uneasiness, but upon search the choreic movements will be found.

Disseminated sclerosis, as has been shown in the chapter on this subject, is not infrequent among children, and the tremor may be confounded with that of chorea; but the choreic movement is, as I have said, twitchy, quickly beginning and quickly ending, or wavy, whilst

the tremor of sclerosis is a continuous series of very fine muscular oscillations; moreover, the nystagmus, the scanning speech, and the other symptoms of sclerosis are entirely wanting in chorea.

The fibrillary tremor of progressive muscular atrophy may be confounded with chorea, but the former is generally a feeling as of a pulse-beat under the flesh, is much more continuous and rapid than the fibrillary movements, and has none of the twitchiness or abruptness of beginning or ending, besides which the atrophy that is present is never observed in chorea.

The tremor of hysteria may be confounded with chorea, but if it be borne in mind that choreic movements do not constitute a tremor, as has been again and again insisted upon, the diagnosis can be readily made, whilst chorea is wanting in the limitation of the field of vision and in the sensory phenomena of hysteria.

Prognosis. Cases of chorea with losses of consciousness that display a tendency to develop are usually dangerous cases, as I have said, as the only two which I have seen terminated fatally; but the cases of chorea with losses of consciousness that show no developmental tendency have the prognosis of ordinary chorea without these losses of consciousness. The cases of chorea beginning after a slight attack of articular rheumatism with rapid pulse and rapid respiration are, if I may judge by my three cases, always fatal. A case of chorea beginning after articular rheumatism of any kind is apt to be more serious than if occurring without it, but most of them recover; indeed, I have never seen a fatal case except where the articular rheumatism had developed cardiac lesions that were themselves fatal. Most cases of chorea which we see are those of the Sydenham or athetoid type, and these have an excellent prognosis so far as death is concerned, but have a serious prognosis so far as relapses are concerned and so far as a tendency may be left to certain awkward gestures and grimaces. Thus of 250 cases of mine, 108 had relapses. These relapses, as I have said, are prone to recur in the spring and the autumn, but they generally recover; *i. e.*, the choreic movements become slight or almost die away, and the *malaise* and disturbed general health of the child disappear.

The chorea of pregnancy has a fatality of about 33 per cent., and is, therefore, a serious form. Chronic chorea is a very intractable form, and cures are infrequent, and this is also the case with electric chorea.

Treatment. The treatment of chorea should be by means of cutting off the expenditure of energy, with good food, arsenic, iron, cimicifuga, cod-liver oil, and electricity.

All cases of chorea occur in children of energetic minds and bodies, so that I am accustomed to say in my lectures that it is rather a back-handed compliment for a child to have chorea, as I never saw a fool of a child afflicted with this disease; indeed, it would seem that all these children are those who are making large drafts upon their reservoir of energy, and certainly the effect of rest upon the disease is proof of this theory. I put all my little patients to bed at once unless the chorea be very slight in type, and even then I

cut off their expenditure of energy to some degree. In most cases I put them to bed for the first week, taking care that their room shall be sunny and properly ventilated and heated. It must be borne in mind that confinement to bed does not mean the same absolute rest for a child that it does for an adult, for the former will at the best be seizing a thousand pretexts for getting out of bed, and will be in constant motion whilst in bed. Yet this relative rest is very important. Moreover, it should be remembered that the repugnance of a child, and generally of the parents, to rest in bed is so great that as much of it as possible should be obtained during the first attack, for it will generally be difficult to obtain it to the same extent in future ones. After I have kept a child in bed for a week and it has begun to improve, I permit it to get up at noonday for the second and third weeks, and then I permit it to rise, say, at ten o'clock in the morning; but during all this time I insist that it shall be put to bed by seven o'clock in the evening. For a month or two more I do not permit the patient to rise until ten o'clock or to stay up later than seven, and during the whole of the period that it is under treatment I forbid walking for any distance, although I may permit the child to take a ride of about an hour in fine weather in the sunny portion of the day. This is about the amount of rest that I should order in an ordinary case of minor type; but, of course, this will have to be far greater in the violent cases or those of severe type. As soon as a child has been put to bed I commence the administration of arsenic, preferably Fowler's solution, in three-drop doses three times daily after meals, in a small tumbler of water, and I increase the dose every two or three days by a drop until there supervenes some slight puffiness of the eyelids, some loss of appetite or disturbance of the stomach, or some distinct paleness of the conjunctiva. Usually these symptoms will appear when the dose has reached seven or eight drops three times a day. I never push it beyond this, because I have satisfied myself that the larger doses of arsenic which have been so much vaunted are not only of no greater effect upon the disease, but that they are often harmful by upsetting the digestion and producing a condition of arsenical cachexia. Some ten years ago I treated all my cases of chorea with large doses of arsenic, giving to some as much as sixty drops in the course of the day, but I found that my results were no better than those that were obtained by Drs. Gray and Tuckwell, of Oxford, England, by simple rest and good food without any arsenic at all, and since that time I have satisfied myself that the full effect of arsenic can be obtained by the smaller doses, and that while the drug has an undoubted effect upon chorea, this is not a lasting one and is not enhanced by toxic doses. When I have reached the dose that produces the slight symptoms of which I have just spoken, I rest contented if the patient has markedly improved; but if I think the patient should improve still more, I then begin the simultaneous administration of iron, using the dialysed iron, thirty to sixty drops three times a day after meals in a cup of water, or Dree's liquor of the albuminate of iron in the same doses, or the peptonate of iron, gr. iij three times daily, in a compressed tablet. The

other forms of iron have not seemed to me to have anything like the effect of these three preparations. I was led to this simultaneous use of iron and arsenic by an accident. I had given one of my patients such large doses of arsenic that arsenical cachexia was very marked, and in order to overcome it I used large doses of iron as an antidote, when to my surprise not only did the arsenicism disappear but the choreic movements rapidly improved. This led to my further use of the two drugs simultaneously, and for several years I was much puzzled to understand how it was that two medicines that were mutually antidotal could have such an excellent effect in combination, until I came across the writings of a German writer demonstrating that iron has a calmative effect upon muscular fibre much the same as arsenic has. With the iron and the arsenic I conduct my patients to the end of the treatment, although in some children cod-liver oil may also be given with advantage in the colder months of the year in our climate if their stomachs will take it without disturbance. I administer the oil preferably pure, a drachm three times daily, after meals; but if the children prefer, I give it in a perfectly fresh emulsion, which should be prepared every week and kept upon ice. It is very rarely that I have met with a patient who cannot take arsenic, but this will happen sometimes, and when it does I use iron either alone or in conjunction with cimicifuga, of which I prefer the fluid extract, ten to fifteen drops three times a day; but a careful trial of the latter in my clinic has shown conclusively that it is not equal to arsenic in controlling chorea. After the patient has gotten over the more acute symptoms, galvanization of the spinal cord with a current of 3 to 5 milliampères every second day at sittings of five minutes each will often be found to be of excelleut effect. (See p. 108.) In some slight cases of chorea I have found that a change of air, either from the city to the mountains or seashore, or *vice versa*, will have a startlingly good effect, so much so that it can often be relied upon to do quite as much as the rest will do. When these little patients are confined to bed I take care, as already said, that the room shall be sunny and well ventilated, and in addition have them take an air-bath every day for an hour in pleasant weather, by having all the doors shut and the windows open, so that they shall be flooded with air without draught, taking care, of course, that they are properly wrapped up. I also take precautions that my patients shall be brought to me every spring and autumn, the moment that any relapse is noticed, and usually these relapses can be treated either by medication of iron, arsenic, cod-liver oil, or cimicifuga, with only slight cutting off of energy, as by keeping them in bed till ten o'clock and sending them to bed at seven, and often without any particular rest at all. I take care also that their idiosyncrasies as regards barometric disturbances shall be studied intelligently, so that if they are disturbed by storms —either in the approach, passage, or after-period—they shall be kept in the house during these times, or, perhaps, put temporarily upon small doses of arsenic. The food of these little patients should be varied and nourishing to the highest degree that is compatible with their age, and I have never seen great benefit result from any special

system of diet. Good milk, eggs, the amount of meat that is suitable to their age, with fruits and vegetables in their proper season, constitute all that is necessary.

Chronic chorea is a very intractable disease, as I have stated, and while I have cured some few cases of it by absolute rest in bed and the administration of arsenic and iron and galvanization of the spinal cord, I have yet seen many cases that relapsed in spite of all treatment. In those cases which I have cured, however, the treatment in bed has been for a period of two or three months, with daily gentle massage and gentle faradization of the whole body so as to prevent the ill effects of this prolonged recumbency. The cases of chorea with rapid pulse and rapid respiration should be kept in bed from the very start, and whilst moderate doses of arsenic are administered, digitalis should also be given, a drop of the fluid extract in a teaspoonful of wild-cherry bark, although as my three cases have terminated fatally I cannot very well say much about the treatment. Electric chorea I have never seen cured, although I have seen it much improved by the same treatment as that which I have spoken of in chronic chorea.

PALMUS (THE TWITCHES).[1]

Synonyms: The Jumpers. Latah. Miryachit. Tic convulsif.

In 1880, Beard, of New York, called attention to a malady that he had observed among the Jumpers of Maine. In 1884, Hammond published an account of an affection known as *miryachit*, a similar disease of Siberia. In 1883, Mr. O'Brien (cited by Gilles de la Tourette) depicted a similar nervous affection of the Malays. In 1885, Gilles de la Tourette published an elaborate description of what was undoubtedly the same affection under the heading of "A Study of a Nervous Affection characterized by Motor Inco-ordination with Echolalie and Coprolalie," meaning by these two latter words a tendency to imitation and to the use of indecent words. Beard found that the Jumpers of Maine did whatever it was told them to do. Thus, one who was sitting in a chair with a knife in his hand was told to throw it, and he threw it so quickly that it stuck in a house opposite; at the same time he repeated the order to throw it with a cry of alarm not unlike that of hysteria or epilepsy. He also threw away his pipe, which he was filling with tobacco, when he was clapped upon the shoulder. Beard rehearsed the first part of Virgil's Ænid and the first part of the first line of Homer's Iliad, with which the illiterate Jumper could not have been familiar, and he repeated the sound of the words as they came to him, in a quick sharp voice, at the same time that he jumped, or threw something, or raised his shoulders, or made some other violent muscular motion. O'Brien

[1] The different names which have been given to this disease, as indicated by the above synonyms, have seemed to me meaningless or un-English; thus, all the cases do not jump, and the name of *jumpers*, therefore, describes a symptom that is frequently absent; *latah* and *miryachit* are Malaysian and Russian names respectively that convey no meaning to the Anglo-Saxon and Latin races, whilst *tic convulsif* or convulsive tic would be confounded in most minds with *tic douloureux*, which has had a time-honored pre-emption of the word "tic." I have, therefore, sought for a new name which should describe the main symptoms, namely,

was conversing with an elderly and respectable Malay woman, and kept up the conversation for about ten minutes without suspecting anything. Suddenly a friend who had accompanied him took off his coat, and immediately the patient began to undress herself, and would have stripped herself completely if he had not interposed, although all the time she was disrobing she was violently angry at the outrage to her sex, calling him an abandoned hog and begging O'Brien to kill him. O'Brien speaks of a cook on a steamer who was carrying his child in his arms one day on a bridge over the river, while at the same time a sailor carried a log of wood in like manner; the sailor threw his log of wood on an awning, and amused himself by causing it to roll over the cloth, and finally let go the cloth, letting the wood fall to the bridge; the cook repeated every motion with his little boy, and killed him on the spot. Gilles de la Tourette shows that these cases have a peculiar motor inco-ordination, consisting of muscular movements somewhat resembling those of chorea, but differing from them in the fact that they are not continuous.

The tendency to repeat words, so-called *echolalie*, and the tendency to indecent conversation, so-called *coprolalie*, are generally attendant symptoms, but not always. In some of these cases there is also a tendency to persistent and fixed ideas, or even a slight delusional condition, with fear of places (agoraphobia) or folly of doubt. Since the writings of Beard, Hammond, O'Brien, and Gilles de la Tourette, a number of cases have been described by other authors, and I have seen several myself. All Tourette's cases (nine in number) began early in life, most of them between eight and nine years, one at fourteen, and one at sixteen.

My own observations have taught me that this disease, studied by the European authors entirely among the hopeless inmates of idiot asylums and in chronic cases, is by no means infrequent in relatively subacute and slighter types in the population at large. I have seen a number of cases among the patients who have come to my office. These are divisible into the following types:

Facial palmus;
General palmus;
Acute palmus;
General palmus with pseudo-melancholia.

Facial palmus constitutes by all odds the most frequent type. It consists of sudden, instantaneous, shock-like movements of lightning-like rapidity, that go to make up a sudden wink or twitch of one cheek, occasionally of both, or of the brow. This series of movements is generally followed by a second series, that, however, is much

the twitching, as distinguished from muscular movement of diseases, with which the malady is likely to be confounded. The word ὁ παλμὸς is a good old Greek word used by Aristotle to describe twitching or palpitation of the heart, and I have simply Latinized it into palmus, which medical men will have no occasion to confound with the Greek word ὁ πάλμυς, "the king," the more especially as the accent is on the first syllable in the latter, whilst, following the Greek derivation, it should be on the last syllable in my Latinized "palmus." The adjective is palmodic, from the Greek πωλμώδης.

weaker, and sometimes even by a third. Sometimes the head is retracted or drawn forward, or one shoulder is elevated. In the majority of the cases the winking is bilateral, but the other movements just named are usually unilateral. I am inclined to think that this is a much more common affection than is generally supposed, or even than could be gathered from my own observation, because I have a strong suspicion that most of the cases of so-called chorea that are treated by oculists are really cases of facial palmus. It is undoubtedly true that in a large proportion of these cases errors of refraction will be found, but what the percentage is I cannot say, because all of my cases have not been examined for this defect, and even if they had been they are not sufficient in number to be satisfactory. But whether this error of refraction is one of the attendant symptoms of the disease, or whether it is a mere coincidence, I cannot undertake to say. I have never yet been able to see in four cases any improvement in the disease result from the mere correction of refraction, although I am inclined to think that in some individuals this correction, together with the use of arsenic, has a beneficial effect. In two cases I have seen a very distinct improvement obtained by the relief of disease of the turbinate bones, but this gain was only temporary in effect. The cases which I have seen of this facial palmus have been of long duration, usually commencing in early childhood and invariably in those whose heredity is neurotic, although not necessarily to a marked degree; in other words, I have seen it occur at the one extreme, as illustrated by a female now under my care who is thirty-one years of age, and whose maternal aunt has been insane, whose sister had a similar trouble, and whose brother had chorea; whilst, on the other hand, I have observed it in several cases of young children, varying from six to ten years of age, in whose family, as would be shown by careful questioning, there had occurred some slight nervous troubles.

The cases of general palmus which I have seen answer very accurately to the description that was given of convulsive tic by La Tourette, except that I have not seen in them any coprolalia, nor the inco-ordination of movement that this author speaks of. They consist of intermittent, sudden, shock-like movements of different muscles of the body. In the face these may be repeated, as in the facial palmus, twice or even three times; but there is not the same tendency to repetition in the other muscles affected. What I suppose would answer to a description of echolalia has also been seen in these cases, although it, I think, is rather sensational and exaggerated to liken it to an echo, for the inarticulate noise is rather a grunting or squeaking sound, very much like that which is heard in many cases of chorea. It has always been accompanied by a sudden movement of the throat, neck, and head muscles, as in a sudden throwing out of a note in singing. It is almost impossible to describe the sound in words; indeed, it has seemed to me more like a sudden spasmodic expiration, such as would be heard if a person were suddenly struck upon the abdomen, than any articulation such as

"*hem*" and "*ah*," as La Tourette would have it to be in his cases. The movement in the legs is usually that of a sudden extension, and in the arms the extensors also seem to be mainly affected. In all of these cases that I have seen the intelligence is not only unimpaired, but it seems to be the rule, as is the case in chorea, that the patients are unusually intelligent; thus, one of my patients, a man thirty-six years of age, who has found this malady a great barrier to his success in life, yet learned stenography in three months sufficiently well to take the prize in a competitive examination into which one hundred and fifty individuals had entered; whilst another patient, the young woman thirty-one years of age, of whom I have already spoken, is of an unusual order of mental capacity. This type is also of long duration, commencing in early childhood and running uncured, so far as I can judge from my limited cases, as far as forty years of age. In one of these cases of this type there were at times exacerbations lasting a week or ten days, in which the patient became exceedingly pale, cold, with slight shivers, and evidencing a condition of widespread motor spasm, in which even the fingers would participate for a few hours, so as to assume the similitude of Raynaud's disease.

I have seen only one case of acute palmus. It was in a boy of six, whose heredity, as far as I could ascertain from the statements of his mother, was not neurotic. He had had trouble some six months before coming to me. He had been labelled with a number of interesting diagnoses, such as chorea, epilepsy, myotonia, hysteria, and neurasthenia. His palmodic movements were very curious. When standing near a table, looking at something, the chin would suddenly come down with a thump that would leave a black-and-blue mark, or his head would be thrown violently to one side, perhaps coming in contact with some adjacent hard object with equal force, or, while standing quietly, his legs would give a sudden twitch, and he would be thrown violently to the ground, and this even happened several times when he was seated on the edge of a stool. The child was under my care for two weeks, and, probably because of an intercurrent attack of diarrhœa, grew steadily worse during that time, in spite of the full doses of arsenic which were administered to him. He was literally covered with bruises from the sudden and violent contacts with articles of furniture, the floor, and the walls. At last, in despair at his condition, I ordered him to be undressed and put to bed, and steadily pushed the Fowler's solution of arsenic until he was taking ten drops three times a day, when to my great surprise he began to improve rapidly, and at the end of six weeks was perfectly well. Keeping him under observation for two weeks longer, I finally sent him to his home in the West, and am informed that he has since remained perfectly well. It has seemed to me that many of the cases recorded as paramyoclonus multiplex have been really acute palmus.

General palmus with pseudo-melancholia. I have seen two cases of this variety. The muscular movements are of the usual sudden, shock-like type, and of the same extent as in what I have ventured

to call the general form. With them, however, there is associated a curious pseudo-melancholia, consisting of certain fixed melancholy suspicious delusions, without, however, any of the suicidal tendencies and abnormal sensations up and down the back of the head, neck, or spine, or the sleeplessness, which are characteristic of most cases of true melancholia. In both of my cases the palmus had existed for a long period, the exact limits of which, however, I could not determine, because the patient scouted the idea that he had had any trouble of the kind, but which the testimony of friends and relatives seemed to vouch for. They were both men, one thirty-six and one thirty-eight years of age. The pseudo-melancholia, however, had only existed in one case for about a year, and in the other for six months. One case passed away from my observation, and I know nothing of its further course. The other case recovered in nine months' treatment, and during the three years that have since elapsed he has been an active business man, although I have not seen him myself during that period, as he took a great dislike to me because I was forced to take strong measures to keep him under treatment, so persistent were his suspicions. One of these cases was mistaken for general paresis, with a melancholy onset, by a competent neurologist of this city, and he persisted in his diagnosis until the course of events demonstrated his error; indeed, it is not wonderful that this should have been the case, because anyone who is not familiar with this type of disease might readily be thus misled. There was, however, no element of dementia in the case; on the contrary, the man's mind, except so far as his melancholy delusions and suspicions were concerned, was keen and intelligent, as were also his perceptions.

The diagnosis of these cases can readily be made. The only disease with which they are likely to be confounded is chorea, from which they differ radically. In chorea the movements are of two varieties, and both usually involve the fibrillæ of muscles and not muscular groups either acting singly or in co-ordination with other muscular groups. The fibrillary movements of chorea, too, consist either of a sudden contraction, quickly beginning and quickly ending, or the contraction in the athetoid form is more gradual. Chorea, moreover, is always generalized, so that the movements are found in the upper and the lower extremities, in the trunk and in the face, and occasionally also in the abdominal and laryngeal muscles. But the movements of facial palmus are always limited to the face; even in general palmus the distinction from chorea is very marked, for in the latter disease the movements are always fibrillary, of the sudden or gradual character that has been described, whereas in the latter they are sudden—shock-like—with much longer intermissions than is the case in chorea.

The prognosis of these cases varies according to the type. I have never yet seen a case of the facial or general variety cured, but I think this statement needs to be taken with some qualification, because in no one of these cases have I ever been able to carry out thoroughly the rigid treatment which is usually so very successful in cases of chorea, namely, prolonged rest, with increasing doses of

arsenic. Whether or not it would be of use in the facial variety might be a matter of doubt; but I believe that there is reason to hope for success in a certain proportion of cases of the generalized type. Then, too, if we carefully study our cases of chorea, it will be seen that there are few of them that are really thoroughly cured, even by any treatment, for the most that we can hope to do is so to diminish the number and the frequency of the fibrillary movements as to obtain a large degree of improvement; but it has been my custom for years to exhibit so-called cured cases of chorea in which patient watching would demonstrate the existence of a few isolated fibrillary twitches. With the relatively weak movements of chorea this is a sufficiently practical result to obtain, but with the sudden, shock-like movements of palmus this would not answer the expectations of patients. The acute palmus was readily cured, as I have already stated. One case of palmus with pseudo-melancholia was cured, I believe, although, as I have already said, I cannot speak positively about this; and the other case I was not able to treat.

There has been nothing to throw any light upon the pathology of this affection. It is true that Gilbert Caddiot and Roger claimed that they stopped a so-called palmus of the ear in a dog by removing the nucleus of the facial nerve in the pons, having without effect previously destroyed the cortical centre of the facial, the internal capsule, the optic thalami, and the cerebellum. But, whilst this experiment is interesting, any neurological experimenter knows that it is impossible to limit a destroying instrument to such a minute space as would be constituted by the facial nucleus.

I am sorry that I cannot say much about the therapeutics of palmus. Arsenic certainly has a beneficial effect upon it, but this is not of so long duration as the same drug has in chorea. My acute case, however, was cured by the arsenic and the rest. It may, of course, be questioned as to how much was done by the rest and how much by the arsenic; but in regard to this I can only say that even before the case was put to bed the arsenic had effected some amelioration. In the facial and general varieties the arsenic alone will not answer, and in the one case of generalized palmus with pseudo-melancholia I could never perceive that arsenic had any effect whatever. Hyoscyamine and hyoscine have a beneficial effect that has only seemed less than that of arsenic. I gave them in doses of $\frac{1}{100}$ grain three or four times a day. Iron, which is so extremely beneficial in chorea in large doses, in conjunction with increasing quantities of arsenic, has been utterly without effect in this disease, as have also the bromides. Galvanic electricity is of distinct benefit, quite as much so as the arsenic in the cases of facial palmus; but I have not had the opportunity of using it in the other varieties. In facial palmus, also, as I have said, I have seen distinctly beneficial results from the removal of errors of refraction or irritative lesions of the naso-pharynx. My impression is that the treatment of these cases should be as follows: Arsenic should be given in the form of Fowler's solution, at first three drops three times a day, and gradually increased, so that in the course of a week or two eight or ten drops three times a day

are taken. At the same time galvanism should be applied carefully to the pes anserinus in facial palmus and to the spinal column in the general form, a current of three to five milliampères being used every day, from three to five minutes at a time. Absolute rest of the patient in bed, for a period of one to two weeks at the outset, should always be used, even in the cases of facial palmus. At the end of this time the patient should be allowed to sit up two or three hours a day, and this period of sitting up should be very gradually increased, so that at the end of three or four weeks more the patient should be out of bed throughout the afternoon hours, but should remain at rest during the morning and evening for a period extending for at least eight to ten weeks from the onset of treatment; and even after this long walks and rides and strenuous occupations should be carefully avoided for several months. All errors of refraction should be corrected and all irritative lesions of the naso-pharynx should be, if possible, removed. In conjunction with these special measures, the patient's general health should be brought to the highest point possible by careful and generous diet and proper tonics.

PARAMYOCLONUS MULTIPLEX.

Paramyoclonus Multiplex is a condition of clonic muscular spasm affecting the limbs and the trunk, occasionally the neck or the face, abdomen or diaphragm. The muscles affected are usually in the trunk and the limbs, and not in the toes and hands; occasionally the movements are tonic as well as clonic; the degree of spasm varies greatly, and may sometimes be so violent as to throw the patient down or out of the chair, whilst in the slighter degrees it can only be observed when the patient is stripped. The muscular contractions are sudden and shot-like, and bilateral; the toes and the fingers are not affected. The symptoms generally yield quickly to treatment, and especially to electricity, so that it is easily diagnosed from chorea, facial spasm, torticollis, palmus, Huntington's chorea, and myotonia congenita.

SALTATORIC SPASM.

This condition was first described by Bamberger in 1859, and is extremely rare. It consists of springing or jumping movements which ensue when the patient attempts to stand, and is apparently due to clonic spasm of the calf-, hip-, and knee-muscles, and in some instances also in the muscles of the back. Its cause is unknown. It yields very readily to treatment, such as general tonics, or copious sweating, and, in some cases, electricity. It is easily differentiated from the jumping which is seen in palmus, because it is a transient disease, and because there are none of the widespread twitchings of palmus, or the imitative tendencies or the mental symptoms of the latter disease.

WRY-NECK.

Wry-neck is due to a shortening of some one of the cervical muscles, generally the sterno-cleido-mastoid. It may be fixed or

spasmodic. In the former variety it is met with chiefly in children, and the causes are obscure. The muscle is often atrophied. In the spasmodic form it usually occurs in adult life, between thirty and fifty-five years of age, and the exciting cause is generally an emotion, or cold, or exposure.

The symptoms are simply those of a malposition of the head from action of some one of the muscles, either the sterno-mastoid, the splenius, or the trapezii. It is usually unilateral. The onset may be sudden or gradual. There may be a certain amount of pain with it,

FIG. 161.

Case of spasm of the trapezius.

but this is seldom acute. Fig. 161 represents a child with spasm of the trapezius.

The pathology is unknown.

The diagnosis is very easy, and all that is needed in order to make it is a sufficient knowledge of anatomy to recognize the position into which the head can be thrown by spasm of the different muscles, as well as sufficient information to be able to exclude any possible co-existing organic disease—which scarcely ever, if ever, occurs.

The prognosis of torticollis is usually good as regards life, but it often is very obstinate, and, in some cases, incurable.

The treatment of spasmodic torticollis by means of belladonna is sometimes beneficial. Leszynsky advises such doses as will be sufficient to cause marked dilatation of the pupil and dryness of the mouth, and I have no doubt that the treatment should never be regarded as ineffectual until this point has been reached, and until the patient has been thus kept under the influence of belladonna for several days or weeks. The cases, however, which I have treated by belladonna have all of them been office patients, many of them living at too great a distance in this city or neighboring cities to permit of my putting them fully under the influence of belladonna, so that my results have been obtained with far less doses. If the case is a

mild one, I usually begin with one drop of a reliable fluid extract of belladonna morning and evening, and increase this to three or four drops, unless the spasm is relieved. Galvanism of the affected muscles is sometimes of considerable temporary value. One pole should be placed on the cervical spine, electrode No. 68 being used, and another pole, electrode No. 71, should be placed over the affected muscles, and a current of three to five milliampères passed from three to five minutes every second day. Faradization of the affected muscle has never proved of any use in my hands. Stretching the nerve-trunk from which filaments come to the affected muscle is often of considerable value. Other drugs, such as morphine, chloral, conium, and Indian hemp, I consider to be almost worthless. If the case should prove to be incurable, and a great deformity has resulted, section of the tendon of the contracted muscles should be resorted to for the relief of the deformity; but it is useless except for this purpose, as it does not check the spasm, and the subjective discomfort of the patient is apt to be greater because the shortened muscle contracts more vigorously. In a hopeless case a mechanical support, so arranged as to give rest to the head, will be palliative.

HYSTERIA.

Definition. Hysteria may be defined as a peculiar increased reflex excitability of the cerebro-spinal nervous system, possibly also of the sympathetic, with decrease of cortical inhibitory power.

Causes. The causes of hysteria are—

- Heredity;
- Diseases of various kinds;
- Trauma;
- Emotion;
- Mental and physical strain;
- Imitation;
- Mental contagion;
- Sex;

Heredity is a frequent cause. It is not necessary that the direct heredity should have been hysterical, for a general neurotic heredity or insanity will suffice. The neuroses and insanities are interconvertible in different generations of human beings, so that it is quite usual to find insanity in one individual or one generation and neurosis in another individual or another generation, or *vice versa*.

Diseases of various kinds are very common causes of hysteria, even in those who are not predisposed, and this fact should always be borne in mind.

Trauma occasionally causes hysteria, it is true; but it has not, in my experience, played the large part in the causation which has been attributed to it by the French school and also by many physicians in this country. (See Chapter on "Railway Injuries," etc.)

The emotions, especially the passions, are frequent causes of hysteria, both in the predisposed and in the non-predisposed.

Mental and physical strain may cause hysteria even in the non-

predisposed, and in the predisposed it plays a much larger part than is recognized by physicians.

Imitation and mental contagion are the causes of those hysterical epidemics which we see occasionally in girls' schools, and of those curious outbreaks of the Middle Ages, of which a history is given in another chapter. (See "Chorea.") Besides this, susceptible children and women, or even men, will become hysterical at times upon witnessing an hysterical outbreak.

Sex. Although females most frequently have hysteria, it yet frequently occurs in males.

Clinical History. The clinical phenomena of hysteria are as various as are the phenomena of the cerebro-spinal and sympathetic nervous systems, and of them may be said what Shakespeare said of Cleopatra (who probably charmed Cæsar by her very hysteria), that

> "Age cannot wither her nor custom stale
> Her infinite variety."

But, various as they are, these clinical phenomena can be classified under the five heads of mental, motor, sensory, visceral, and convulsive symptoms.

The typical mental symptoms of hysteria are generally supposed to be an alternation of laughing and crying, with an accompanying *globus hystericus*—*i. e.*, a feeling as if something were in the throat; and it is generally supposed that the diagnosis must be based upon them. This is a very serious mistake, for, as a matter of experience, it is only a very small proportion of cases in which these symptoms are observed. In reality, the mental symptoms of hysteria in the slighter degrees are practically undistinguishable from those of caprice or lack of self-control, whilst in the graver forms they equal in severity the mental disturbances of such neuroses as epilepsy or catalepsy, such cerebral alteration as is indicated by the different forms of coma, or such as is present in those forms of insanity to which has been given the name of the "psycho-neuroses." It is the rule that the graver forms manifest an altered mental condition only at the time of the attacks, whilst milder types manifest a far more continuous abnormal mental condition. When the peculiar mental condition is observed in the milder forms, it consists of a tendency to an emotional condition that is not controlled by the usual cortical inhibition; or, to put it in less technical language, the emotions and the passions and the sensations have freer play than is usual with an individual of the same sex and condition in whom self-restraint, a proper sense of the fitting, and good judgment act as guides. An hysterical individual can be made to laugh or cry, can be angry, or depressed, or elated, or frightened, without adequate cause in the environment, in the circumstances, or in the condition of the health otherwise. In the typical cases of hysteria of milder degree this non-inhibited condition of emotionalism is often pitiable; as I have seen it in a woman who, having passed through several attacks of insanity caused by terrible domestic misfortunes and attempts at suicide, could not be spoken to upon any subject that

required ordinary thought, could not hear the noise of a door closing or a door-bell ringing or a church-bell chiming, or could not discuss some ordinary matter of domestic detail, without having her eyes fill with tears and her face twitch convulsively. In the severer types of mental disturbance the mental symptoms approximate those of insanity or coma. An hysterical patient who is violently excited is, for the time being, often as far beyond control as a case of acute mania, and the mental confusion is often quite as great. In these violent outbreaks the salient phenomena are the violent emotionalism, the incoherence, the impulsive acts, and the entire loss of mental inhibition. I have seen many curious and almost incredible exhibitions of this kind. I have seen gentle women, ordinarily of mild temper and perfect modesty, behave in a way that would have shocked a female of the savage tribes of equatorial Africa, and that did overwhelm them with mortification when the attack had passed. I have seen ordinarily strong and self-controlled men, holding positions of trust that called for the constant exercise of great decision and good judgment, lose such control of themselves as to behave like panic-stricken boys. The same mental characteristics obtain in hysterical insanity. (*Vide* " Mania.") After most of the violent outbreaks—indeed, during the occurrence of them—there is a varying degree of mental confusion that not infrequently passes into partial or entire unconsciousness, so that the patient may not afterward remember what has taken place.

The sensory symptoms of hysteria are characterized by the same capricious element that is observed in the mental condition, so that they are prone to come suddenly, leave suddenly, and to persist for most uncertain lengths of time. The sensory symptoms are infinite in character and variety, being sometimes referable to the motor nerves, sometimes to the sensory nerves, sometimes to the sympathetic nervous system, and ranging over all the tissues of the body. An attempt has been made by Charcot and his followers to establish the dictum that the sensory symptoms of hysteria are generally those to which they have given the name of hemianæsthesia, and in which, as the name indicates, there is an impairment of the different sensory nerves upon one side of the body in varying degree, with the single exception of the optic nerve, which is usually affected bilaterally. This optic nerve, it is claimed, shows its implication by a concentric limitation of the visual field and by impairment of the color-sense, whilst the olfactory, the gustatory, and the cerebro-spinal nerves of tact, pain, temperature, and the muscular sense are impaired in the usual way. It is not, however, claimed that this hemianæsthesia is usually a complete one, inasmuch as the concentric limitation of the field of vision, with some impairment of the color-sense, hyperæsthesia, slight analgesia, and slight impairment of the tactile sense, constitute the symptoms of most cases. Coincident with these symptoms, according to the French school, is a tenderness upon pressure of one or both ovaries. But this Parisian dictum has not been confirmed by other neurologists. Thompson and Oppenheim have shown that hemianæsthesia, in the sense defined, is observed

in very many other diseases than hysteria (*vide* "Railway Injuries," etc.), and, if I may trust my own experience, in this country it is an infrequent symptom. It is possible, however, that too little account has been taken of the inherent differences that there are between the Latin, the Anglo-Saxon, and the race that is growing up in this country from a larger fusion of different nationalities than has ever taken place before in the recorded history of the world.

The visceral sensations, which are only, of course, a subdivision of the sensory, are as infinite in variety as the latter. The most familiar one is the globus hystericus, that curious muscular sensation of the larynx; and, indeed, the hysterical are exceedingly prone to abnormal laryngeal and tracheal sensations of various kinds, from the typical aphonia to the obstinate difficulties of deglutition which so often puzzle laryngologists. The so-called ovarian tenderness is probably merely a muscular or visceral sensation, or hyperæsthesia, because in many cases it extends over the whole flank, or, indeed, the whole side, and its analogue in the male is found in a similar sensitiveness of the testicle. Support is lent to this view by the grave doubt that has been cast upon its being possible to reach the ovary in the precise locality which is indicated by Charcot. Borborygmi and flatulence are other very common forms of perverted visceral sensations in the hysterical. It is not uncommon to witness as rigid a distention of the anterior abdominal walls as is seen in cases of peritonitis, and, indeed, the differential diagnosis may sometimes be a matter of difficulty. Hysterical dyspepsia is sometimes observed, although it is rare. The secretion of large quantities of limpid urine in which the solid materials are not notably increased belongs to the same class of phenomena.

The motor disturbances of hysteria commonly accompany the sensory ones; but the so-called hysterical joint is an example of motor disturbance pure and simple. Attention was first called to it by Sir Benjamin Brodie, and it has been well studied by the Schleswig-Holstein surgeon, Von Esmarch. It may be bilateral or unilateral, and is not attended by any of the swelling, redness, or great painfulness of the joint seen in organic joint troubles. The joint of the knee is usually affected. I have seen this, too, in some rare instances after the cure of a melancholia.

The convulsions of hysteria can be divided into two classes—the purely hysterical and the hystero-epileptic. In the former the loss of consciousness is seldom complete, so that the patients never fall in such a way as to injure themselves as they do in epilepsy; whilst the convulsive movements of the extremities, instead of being characterized by the limited range and automatic character of the tonic and convulsive movements of epilepsy, are of much wider range and are much more purposive. In epilepsy, for instance, the muscular movements usually consist either of muscular rigidity, with or without slight tremor, or of shock-like alternate movements of flexion and extension. In hysteria the patients throw the limbs about, or roll over, or strike out, or kick. Charcot has made very precise studies of hystero-epilepsy, which he also calls *hysteria* major, and

an attack of which he divides into four periods, as well as a prodromal stage. The latter consists of *malaise*, anorexia, occasional vomiting, silence, and melancholy, or excitement, increase of a pre-existing hemianæsthesia, visual hallucinations, globus, etc. The first period is the epileptoid, which is subdivided into the tonic and the clonic stages, and that of resolution. The second period is that of contortion, in which the patient has widespread and half-purposive movements, or a series of salaaming movements. The third period is that of hallucinatory or melancholy attitudes which are evidently the result of passing mental changes. In the period of resolution the patient manifests either a melancholy delirium with hallucinations of animals, or general or partial contractures, which are often painful, or great exhaustion. The attack generally lasts about a quarter of an hour, and, according to Charcot, it can be stopped by pressure upon the ovarian region, or by an electric current. After it has ceased the patient may remain in a similar condition to that of *status epilepticus* (*i. e.*, comatose) for as long a period as twenty-four hours. But this hysteria major is seldom seen either in this country or in England; indeed, I have never myself seen more than a dozen cases of it in an experience of twenty years.

Prognosis. The prognosis of hysteria varies greatly. The hereditary forms are seldom or never entirely cured, although careful attention to the general health and to the removal of exciting causes may effect immense improvement. The attacks occurring in the non-predisposed, or the hysterical status which may be excited by various causes in such individuals, are always perfectly curable.

Diagnosis. At the outset of this aspect of the subject, let me seriously impress upon every medical man the need of being very cautious in making a diagnosis of hysteria. For ages it has been too much the custom of the medical profession to ascribe unknown diseases or the effect of undiagnosed diseases to hysteria. I have been present at a number of autopsies of individuals supposed to have been hysterical, but in whom a gross organic lesion has been found: in several cases, an aneurism; in two, a nephritis; in one, a myelitis; in one, a pachymeningitic hemorrhage. It has been my fortune, too, to be called in consultation a number of times to so-called hysterical cases really suffering from organic diseases, such as intracranial syphilis, myelitis of the anterior spinal cornua, progressive muscular atrophy, neuritis, diabetes, pelvic abscess, etc. Even in those who are known to be predisposed it should not be forgotten that the immediate exciting cause of hysteria may be an organic lesion. In any doubtful case, therefore, the urine should be carefully examined quantitatively, qualitatively, and microscopically; the heart and lungs should be carefully auscultated and percussed; the ovaries, the Fallopian tubes, and the uterus should be carefully examined, and all possible diseases should be thought of. In fact, the diagnosis of hysteria calls for the exercise of the broadest information and the greatest skill. The history of the patient should be patiently and carefully elicited. Especially should the following nervous diseases be excluded:

Neurasthenia;
Melancholia;
The early stages of other insanities;
Progressive muscular atrophy;
Lumbago, especially that following stricture;
Hydrophobia;
Simulation.

Do not forget that neurasthenia may be an exciting cause of hysteria, so that the latter may complicate the former and sometimes render it a difficult matter to know how much is due to the one disease and how much to the other. In true neurasthenia, however (*vide* section upon "Neurasthenia"), the emotional condition, the aberrant and capricious motor and sensory phenomena, and the convulsive symptoms of hysteria are lacking. The *facies*, too, is different; the hysterical individual does not have the worn and anxious countenance of the neurasthenic. In the latter, too, the narration of the symptoms is made precisely, without exaggeration or emotion. In lithæmic neurasthenia the feeling of fulness and tension about the head, the irritability, the vertigo, the numbness and tingling, and the other paræsthesiæ are more constant in duration and location than similar symptoms would be in hysteria. In traumatic neurasthenia the history of the trauma, the melancholy that is often in marked contrast to the self-control of the patient in other particulars, the spinal ache, the slight insomnia, the frequent neuralgia, persistent in one or more nerve-trunks, the muscular atrophy, the occasional paralysis of a group of muscles or a limb, the peculiar muscular movements—all constitute a symptom-group that is lacking in the excessive emotional condition and the seeming caprice and variability of the phenomena that are characteristic of hysteria (*vide* "Railway Injuries," etc.).

Melancholia is always attended, as I have pointed out (*vide* "Melancholia"), by two or three of the three symptoms, viz., melancholia, the post-cervical ache, and the insomnia; and even if these are not sufficient, the sad and suspicious *facies*, the hallucinations, illusions or delusions, the suicidal impulse, the dull cerebral reflex, or the melancholic agitation, are amply sufficient signs for a differential diagnosis.

Commencing subacute mania (*vide* "Mania"), or the early stage of general paresis (*vide* "Paretic Dementia"), is sometimes mistaken for hysteria. In subacute mania, however, the accelerated mental state, the quickened cerebral reflexes, the mental and physical restlessness, the heavy countenance that contrasts so sharply with the seeming brightness of retort to an individual question, and the slight mental confusion, are symptoms that are not seen in any form of hysteria except hysterical mania. General paresis in its early stages is sometimes characterized by so much irrationality of conduct and action as to cause the patient to be suspected of caprice; but the tremor of the tongue and facial muscles, or even of the body, the inequality of the pupils, the paroxysms of facial flushing, unsteadiness of gait, and irrational conduct can almost always be elicited by careful examination or questioning; whilst the diagnosis is easy in the

more advanced stages when the dementia, stupid delusions—usually of grandeur—general tremor, and shuffling gait have supervened.

Progressive muscular atrophy may be overlooked in some instances where weakness has been complained of, or in the cases that are probably due to peripheral neuritis, wherein complaint has been made of the pains. In any suspected cases the patient should be stripped, and a careful examination made of the different muscular groups (*vide* "Progressive Muscular Atrophy").

Lumbago is not infrequently very persistent and very intractable to treatment, so that the failure to relieve the patient may be ascribed to hysteria. But the lumbago following stricture is such a curiously intractable complaint that there is a special danger of confounding it with hysteria (*vide* Chapter on "Neuralgia").

That the symptoms of hydrophobia may be purely hysterical is undoubted. In such cases, however, there is a lack of the prodromal stage of hydrophobia; and strong assurances of safety, with isolation, will speedily make the matter plain. In the interests of science, no injection of the Pasteur virus should ever be made until hysteria has been excluded, for in an hysterical case the injection of cold water would probably answer quite as well. In doubtful instances, however, where danger seems imminent, the patient should be given the benefit of the doubt, and the proper injection of Pasteur's virus should be made (*vide* "Hydrophobia").

Treatment. The treatment of hysteria will call for the exercise of much intelligence, tact, and knowledge of human nature, as well as of medicine. Before entering upon the task the physician should disabuse his mind entirely of the idea that hysteria is a caprice, and not a disease, and should thoroughly imbue himself with the further idea that although the hysterical individual is exceedingly impressionable, he or she may yet have the other diseases to which the human body is subject. The brutal and forcible methods of former days should never be thought of for a moment. The patient should be thoroughly studied, and all the circumstances of environment should be carefully inquired into. For purposes of treatment hysterical individuals may be divided into two great classes: those who are suffering from hysteria alone, and those who are afflicted with hysteria complicated by other diseases. Those who are suffering from hysteria alone should be treated somewhat differently accordingly as the hysteria is mild or severe. In the mild cases the confidence of the patient should be gained by the infinite devices that will suggest themselves to a physician of experience and knowledge of human nature. When this has been done, the cure is often effected with surprising facility, and it is a matter of little consequence what particular remedial measures are employed. I have had many young men and girls brought to me who were suffering from slight symptoms that their hysterical nature caused them to magnify into enormous ones, so as to produce exactly the same psychical effect as if they were weighted down with organic disease. The boys have abandoned their careers perhaps, and, aided by the anxiety of well-meaning relatives who could not be made to understand the true

nature of their symptoms, they have wearied of physicians and resigned themselves to what they supposed was their destiny of a lifelong valetudinarianism, overshadowed by vague forebodings of impending evil. Girls are more prone to fall into a similar hopeless condition, not only because of the greater impressionability of the female mind, but also because of their less tendency to struggle against adverse circumstances. Yet I have on my case-book the histories of many such patients whom I have succeeded in restoring to energetic participation in the struggles of life without the use of other measures than gaining their confidence, gradually and firmly leading them to exert their self-control, aiding them in this task, accustoming them to understand that their sensations, however alarming they may seem, are really of no importance, and without the use of drugs further than as placebos. The example of the quack through all ages has been too much contemned, I think, by the regular practitioners of medicine, and yet every physician of experience knows of the startling successes that they occasionally obtain. Indeed, I believe that if hysteria were to die out from the human race the fraudulent practitioners of medicine would be more than half exterminated. Nevertheless, impostors as they are, these men have done with fraudulent intent what the scientists ought to do with an honest purpose, namely, recognize that the human mind is a much more potent instrument to play upon than the nervous system of the stomach and intestines. I deal very frankly with this class of patients. I tell them that their disease is a purely functional one, and that it can be perfectly cured. Then, bit by bit, I teach them that they can do many things that they had thought impossible, taking a special care never to have them enter upon a task that is too great for them to conduct to a successful issue, because a failure with an hysteric is apt to be fatal. By this means I gain the prestige of success with them, and each succeeding task in entered upon with a feeling of greater confidence. Sometimes I talk kindly, sometimes I speak very sharply; but through it all, whether I sympathize with them or whether I reprove them, I maintain their confidence. For example, a girl is brought to me who cannot go among crowds. I gradually get her accustomed to them. A boy cannot take part in athletic sports. After having gained his confidence, I put him in charge of some one versed in athletic sports who has good judgment, and whom I instruct to get him to enter into them by degrees. In the severer cases of hysteria, however, the patient must be removed from an unfavorable environment, taken away from home and friends, and put in charge of a trained nurse who is possessed of good judgment and tact. The selection of this nurse is a matter of considerable importance. It would be a fatal mistake to place an old and worn-out woman in charge of a young and active girl; or an ill-bred, coarse, untidy woman in charge of one who is refined and particular about her person and dress; or an unkempt, frowzy, ill-conducted, ill-mannered man in charge of an educated and well-bred gentleman. If the patient suffering with hysteria is one of that unhappy type

of bedridden individuals of whom neurologists know so many, the nurse should also be a woman of firmness. Even in these severe cases, however, the same method of psychical treatment should be employed. It is supremely ridiculous at times to witness the rudimentary education that must be impressed, detail by detail, upon these hysterical individuals, with an unflinching persistence of purpose. I have put upon their feet and practically cured patients who had been bedridden for thirty years by means of rigid attention to these minutiæ. Day by day they must be taught like a little child to do some one thing that they had not done the day before, until, as the weeks creep by, the accretion of these little things have put them on their feet. First, they are told to stand for five minutes each day, then for ten minutes, then for fifteen, and so on. Next, they are told to take a walk around the room. Next, as mental control over them is gained, they are suddenly some fine day told to go out-doors. The heart must, of course, be carefully watched in these patients who have been bedridden so long, and if it grows weak and rapid after some exertion of this kind, it is often advisable to begin the administration of $\frac{1}{25}$ grain of the sulphate of strychnine three times a day, either in the form of pill or tablet triturate. If there is anæmia conjoined with one of these severe cases of hysteria, the treatment by rest, large quantities of food, and iron should be instituted, as I have advocated it in the chapter upon "Neurasthenia." All other sources of bodily discomfort should be sedulously attended to. True hysterical convulsions need no special treatment further than the exhibition of some one of the well-known antispasmodics and sedatives, such as the nitrite of amyl in the so-called "pearls" containing three to five drops, one of which should be broken in a handkerchief and placed under the nose of the patient; a dose of bromide of potash, gr. 15 to 20; the inhalation of a small quantity of ether or chloroform; a teaspoonful of Hoffmann's anodyne in a wineglass of water; or sometimes a simple hypodermic of cold water. I have, however, again and again seen these convulsions brought to an end by firm and judicious psychical treatment and isolation. Hystero-epilepsy should be treated as indicated in the chapter upon "Epilepsy." Hemianæsthesia occurring in hysterical individuals should be treated by a combination of psychical measures and electricity, the galvanic current being applied to the spinal column, and the faradic current brushed over the affected limbs with two wire brushes, the current being strong enough to be distinctly felt by the patient. It should always be remembered, however, that hysterical individuals are not exempt from the ills that flesh is heir to, and that they may just as likely have neuralgia, neuritis, neurasthenia, etc., as individuals who are not hysterical, although they are apt to respond much more promptly to treatment than the latter.

To sum up, therefore, the treatment of hysteria should be mainly by means of psychical measures. If, however, other diseases complicate the hysteria, these should also be carefully looked after.

HYPOCHONDRIA.

Hypochondria is a pseudo-mental disease that can scarcely be called an insanity, consisting of a tendency to morbidly exaggerate the various sensations of the body and their importance. At times this exaggeration is so great as to amount to actual delusion. Every physician is familiar with these cases, for they are the bane of every medical man. They complain of all sorts of sensations and dwell upon and recapitulate them *ad nauseam*. One patient thinks that his hair is falling out; another that he is growing weak in his muscles; another that he has this, that, or the other symptom. They all take medicines as a steady diet, and they all fall a ready prey to quacks and every "ism" that the morbid ingenuity of man can suggest. Hypochondria is sometimes met with in conjunction with mental disease; and in some cases of mental disease there may be a prodromal period of hypochondria, and a hypochondriacal insanity has even been described, but this latter is certainly rare. Sexual hypochondria—that is, hypochondria about sexual matters—is a very frequent form, and is often described under the name of sexual neurasthenia.

The diagnosis of hypochondria is usually very easy. The only questions that can arise are as to whether the patient is not suffering from melancholia, or the prodromal stage of some insanity, or from some actual coexisting disease. From the melancholiac the hypochondriac can be readily distinguished from the fact that the melancholiac does not seek relief, is not prone to believe that medicine can afford him any aid; and in the slighter forms he also suffers from the symptoms to which I have elsewhere called attention ("Melancholia"), namely, insomnia, post-cervical ache, and the suicidal tendency; besides which he has a peculiar *facies* of his own that a little experience will soon make familiar; whereas the hypochondriac readily takes medicine—even eagerly solicits it—has none of the three cardinal symptoms that have been mentioned above, and has no peculiar *facies*. The hypochondriac prodromal stage of an insanity is usually not prolonged over a few days or a week, and this fact alone would distinguish it from the chronic course of hypochondria, which is often life-long, even if the succeeding mental symptoms of the insanity were not sufficient. Any disease that may coexist with insanity must, of course, be recognized by the general medical knowledge of the physician, and it is useless to catalogue these diseases, as they embrace almost the whole field of medicine, especially in the adult.

The treatment of hypochondria is very difficult. I believe that the most success is to be obtained from the principle of suggestion, that can only be fully understood after reading the chapter upon "Hypnotism;" and, as I have cured several cases of my own by this method, I can give full credence to the many cures that have been reported by the French schools of hypnotism of Nancy and Paris. It is not always necessary by any means to induce full hypnotism. It may be well sometimes in obstinate cases to hypnotize the person sufficiently to prevent him from opening the eyelids, but in very many cases even this is not necessary, and it will suffice if the con-

fidence of the patient is gained by making quiet and dogmatic assertions and ordering placebos. Any person, however, who adopts this method of suggestion must be fully acquainted with the hypnotic methods, so as to be prepared to carry out the treatment thoroughly. I do not mean to be understood as saying that full hypnotization of the patient would not be the best, but I merely mean to advise that it is not usually politic to use the word hypnotism to the patient, or to let him suppose that hypnotic methods are being used, as there is a widespread prejudice in the lay mind against hypnotism, which is generally thought to be synonymous with quackery, and anything that disturbs the confidence of the patient in the medical attendant will be fatal to the treatment.

Occupation of any kind that will divert the patient's thoughts, or travel, or change of scene, or great emotions, will often act as temporary palliatives and sometimes as cures, and therefore it may be well to make use of these methods. Of course, it is not always easy to evoke an emotion, but this can be sometimes done. In one case of mine, for instance (the patient elsewhere referred to as coming to me with a specimen of black semen), a cure was effected in this manner: I requested him to look sharply at the piece of rag upon which he claimed there was black semen. I then took a black-bound book and laid it alongside of this piece of rag, and asked him if he thought the book was of the same color as the stained rag. He said it was. I then told him that if he thought the rag stained a faint yellow was of the same color as the black book-binding he must be suffering from one of two troubles. In the first place, he might have a delusion, but that involved insanity, and I had treated him long enough to know that he was not insane. The other was that he was a fool, and I bluntly told him that that was my diagnosis. He, naturally enough, started back in anger, and after some angry remarks, left my office; but he came back in the course of a week or two to tell me that he believed I was right—that my blunt remark had made him realize how foolish he was; and since that time—eleven years having now elapsed—he has manifested but very slight symptoms of hypochondria. I do not tell this as a funny story, but simply to show that emotions can be evoked at times by the ingenuity of a physician. Indeed, I believe that in the capacity to play upon the mind in its various functions, especially those which we now call emotions, the future therapeutics of hypnotism will lie; for at present we are stupid enough to think that it is science to play upon the peripheral terminations of the nerves or the structures in which they terminate, whilst we deem it quackery to make use of the gray matter and all its wondrous molecular play. So-called faith-cure, "Christian science," the great emotions of wars and politics, and love and anger, and ambition and avarice, and, finally, the wonderful but only half understood phenomena of hypnotism, are all neglected clinical illustrations of the magnificent influence of the mind over the body; and in the full day of medicine, into whose dawn we are now peering, we shall make proper application, I have no doubt, of these therapeutic methods.

NEURASTHENIA.

Synonym: Nervous prostration.

HISTORY. Functional diseases of the nervous system have been held largely in contempt unless they were fortunately accompanied by such startling phenomena as loss of consciousness, convulsions, epilepsy, the pain of neuralgia, or the often violent muscular contractions of chorea. The medical world has no heredity of training in nervous diseases. For centuries medical writers and teachers have called attention to the phenomena that could be studied in the pulse, in the heart, in the tongue, in the feces, in the temperature of the body; and within a few generations these and a few kindred details have been made the language of a more precise diagnosis of thoracic and abdominal diseases. But nervous diseases constitute a modern study. We may date them back really to the fifties, when the great French school began its career; and it is only within a very few years that the medical schools of the United States have even thought of having a chair especially devoted to this branch of medicine. So it is that while tens of thousands will be prompt in detecting the first signs of deviation from the normal in the pulse-beat, in the respiration, in the tongue, in the *facies*, in the urine or feces, there are scarcely ten—and these only with some special education—who will recognize the primary symptoms of organic or functional nervous disease, which they are only too ready to ascribe to hysteria. Yet all attentive observers of disease, more especially those engaged in neurological study, could not help observing many phenomena that could not be classified; and this, I think, is the explanation of the enormous success that the writings of Dr. George M. Beard upon neurasthenia have had in Germany—indeed, it may be affirmed that he has started a literature in that country, the contributors to which are such well-known writers as Arndt, Krafft-Ebing, Erb, Westphal, Moebius, Seeligmüller, Strümpell, and Langstein. As far back as 1828 C. Brown, of Glasgow, had described what he called spinal irritation, and in 1860 Bouchet had written fully upon what he termed *l'état nerveux;* each of these writers has had his followers to some extent, but Beard, it may be said, was the first to formulate boldly the proposition that there was a widespread condition of functional nervous disease, for which he proposed the name of "neurasthenia" and for whose maintenance and prevalence and varied phenomena he battled with the most alluring and picturesque of pens. It is curious, nevertheless, that the Germans themselves do not seem to admit that the malady is frequent among them, but always fall back upon Beard's assertion that America is its home. In 1874 Murchison, in his brilliant course of Croonian Lectures, delivered before the Royal College of Physicians, described, under the heading of "Functional Diseases of the Liver," a group of symptoms for which he suggested the name of lithæmia. For the same condition Austin Flint had some six years before coined the name of uricæmia; but Murchison's designation has been the one that has come into vogue in the time which has since elapsed. Since

then contributions to the subject have been made by Da Costa, McBride, Lyman, Hudson, Potter, and myself. The general feeling, in this country at least, is, I think, that the term neurasthenia, as defined by the writers upon the subject, embraces entirely too much. I share in that feeling myself. The fact seems to have been forgotten that there are already a number of well-studied functional nervous diseases, as well as that each one of these has symptoms correlated with it, and that no scientific advance is made by jumbling together the symptoms of these various and differently acting diseases under one wide name. Then, too, it should not be forgotten that the nervous system stands pre-eminently alone among the tissues of the body in the highly important fact that its ramifications carry its influences into every other tissue, and in turn carry back to it the influences of every other tissue, so that, much more than is the case with any other organ, the nervous system is peculiarly at the mercy of reflex disturbances. Excluding, then, vicious habits, reflexes from non-nervous organs, and well-recognized functional diseases, the question arises: Is there such a thing as nervous weakness or neurasthenia? I believe there is. But I also believe that it is much more limited in its relative prevalence than is maintained by the eager followers of Beard.

Clinical History. There are, in my opinion, three forms of neurasthenia:

1. Reflex neurasthenia;
2. So-called lithæmic neurasthenia;
3. Simple neurasthenia.

1. The neurasthenia that results by reflex from non-nervous organs and vicious habits is, in my experience, the second most frequent form, and can often only be recognized by a very careful examination of the different organs of the body. The very essence of a reflex disturbance is its caprice, so that no one set of symptoms can be relied upon, as a rule, to be indicative of a pure reflex. There are, to be sure, some seeming exceptions to this statement, as in the occipital pain of nephritis, the uneasiness at the vertex in uterine diseases, etc. But clinical experience has taught me that these vaunted localizations of pain are all of very little practical value. I know of no shorter way of determining whether a reflex disturbance in a non-nervous organ is the cause of a given neurasthenia than by an examination of the different organs, especially the kidneys, the eyes, the naso-pharynx, the lungs, the heart, the liver, and the genitalia, and in every case the habits should also be carefully inquired into.

2. *Lithæmic neurasthenia.* This form is the most frequent of all, I think, and its symptoms are vertigo, a sense of pressure about the head, slight insomnia, nervousness, occasional susceptibility to odors, occasional tinnitus aurium, neuralgia, paræsthesia, rarely slight anæsthesia, muscular cramp and twitchings, and vasomotor disturbances. It has also been stated by some of the authors that in this type we may also have fever, a delusional mental condition, even myelitis of the anterior horns, epilepsy, and disseminated sclerosis;

and when I wrote my first paper upon the subject in 1886 I was inclined to think that there was a grain of truth in these statements; but an experience since of nine years, ranging over several hundred patients, has failed to bring to my observation a single undoubted case with these symptoms, and I think the mistake has lain in overlooking the fact that the diseases with these symptoms may be associated with lithæmic neurasthenia.

The vertigo is both subjective and objective, although it is generally of the latter kind—that is, surrounding objects seem to be in motion and not the patient; or the patient may reel suddenly when walking or when sitting; or he may feel a sudden vertiginous sensation, the latter often so pronounced as to simulate the vertigo of Ménière's disease. The vertigo is usually of short duration, although it generally recurs frequently, and in some exceptional cases, especially during the hotter months of the year in our climate, is so continuous as absolutely to incapacitate the person from an active life, whilst the moral effect is occasionally incredibly depressing. Thus I have known prosperous and ambitious careers to be absolutely checked by it for the time being; and many a man have I seen go forlornly from clime to clime in search of relative safety, weighed down by the calamity of a supposed incurable malady which a short course of intelligent treatment would have done more to ameliorate than the soft airs of the Hebrides or the mountain grandeurs of Switzerland. Indeed, I know at this present time of one gentleman who has entirely abandoned an excellent business and hopelessly buried himself on a small farm; of another well-known public man whom an attack of this kind of dizziness caused to forsake abruptly his official post and travel hundreds of miles home in a special train with the idea that he was about to be seriously ill; and I know of still another public man of national reputation whose political aspirations have been held in check for a decade by a needless fear of this symptom. This vertigo may come without the slightest apparent reason, or it may have many exciting causes, such as fatigue, mental or physical, special articles of food, often varying in each individual, sometimes a few glasses of hock or a single glass of burgundy or champagne; tobacco, especially cigarettes; or slight strain of the ocular mechanism of accommodation. Purgatives and salines, perhaps relieving the other phenomena, may increase the vertigo, although this increase is usually temporary unless the purgation is extreme or is continued so long as to depress the general nervous system. It can usually be palliated by lying down.

A sensation of fulness about the head is a very constant symptom, but it is not a true headache, although at certain periods of exacerbation it may become such.

According to Da Costa and Murchison, repeated vertigo may lead to a limitation of the visual field, amblyopia, and double vision. I used to think that this was true, but a further and more accurate knowledge of the malady has taught me that ocular conditions of this kind are due either to hysteria or to some organic disease of the nervous system.

I have never seen the temporary blindness of one eye or part of one eye or the retinal congestion described by Da Costa. The last condition is, as is well known to ophthalmologists, often difficult to determine, and it may well be that what one observer may regard as hyperæmia will seem dubious to another.

Nervousness, next to the vertigo, is the most frequent of all the symptoms, and it may be accompanied by irritability of temper, and both these symptoms will often be beyond the control of those who have usually a strong will, so that they are more apt to be present in those of normally feeble volition, when the effect is often mistaken for the cause and hysteria is diagnosed.

The tinnitus aurium is a simple roaring of varying degree, usually without any attendant aural or naso-pharyngeal trouble, although it may be somewhat aggravated by naso-pharyngitis obstructing the orifice of the Eustachian tube, and in gouty subjects Sir James Paget has described a peculiar reddish and glazed appearance of the fauces, as if they were smeared over with glycerin.

The insomnia is usually very slight, and must never be confounded with the insomnia of simple melancholia, which, as I have elsewhere shown, is accompanied by melancholia of a peculiar type and post-cervical ache, and is often mistaken for neurasthenia.

There is an occasional morbid susceptibility to odors, and I have known patients to grow pale and nauseated at odors, or indeed savors, that would ordinarily be unnoticed by them.

The most usual forms of neuralgia are sciatica, neuralgia of the different nerves of the arm and forearm, of the tongue, of the breast, intercostal neuralgia, gastralgia, and enteralgia. The fifth pair is seldom affected. Da Costa believes that a neuritis is occasionally seen, but I have never seen one—indeed, I am of the opinion that the absence of neuritis is a diagnostic sign of value in differentiating lithæmic pains. There are certain pseudo-neuralgias consisting of vague aching pains like the so-called "rheumatic" ones, viz., a sharp pain in the eyeballs, dull pains in the ensiform cartilage, a dragging sensation along the course of the ureters, slight wandering pains which may be mistaken for the ill-defined ones of locomotor ataxia, vague distress over the region of the liver, etc.

Paræsthesias are very frequent, and consist of numbness, tingling, fuzziness of the body or extremities, and a whirling sensation at the crown of the head.

Slight anæsthesias are occasionally, but rarely, observed.

Cramps generally affect the gastrocnemii, usually at night and in cold and damp weather. Twitchings of the face and eyelids are occasionally observed, or even choreic movements in children, and, most rarely of all, fibrillary muscular contractions.

Vasomotor disturbances are among the most frequent symptoms, such as flushings and pallor of the face and sensations of heat and cold, and McBride has described that rare lithæmic form of vaso-motor spasm of the upper extremities known as "digiti mortui."

The typical urine of lithæmics has the following characteristics: It is acid, highly colored. It possesses a high specific gravity, which

is usually 1.028 to 1.034 in a morning specimen, and it may be 1.030 in the whole amount passed in the twenty-four hours. As to the quantity, McBride and Da Costa differ. The former says that it may be normal, increased, or diminished, while the latter affirms that it is usually very scanty. My experience coincides with McBride's. Uric-acid crystals are often visible to the naked eye, in the well-known "cayenne pepper" forms. The pigment is increased. Microscopically, uric-acid crystals are seen, occasionally octahedral crystals of oxalate of lime, commonly the amorphous urates, globular crystals of urate of sodium with spicules. The urea is not generally increased when flesh is being lost (Da Costa). I have recently been able to demonstrate that most of these cases will have small amounts of albumin and sugar in the urine, and also a few hyaline casts; thus evidencing a marked tendency to increase of the nitrogenous excretion. It also is possible that the hydrocarbons passing off by the lungs would also be greater than usual. There is no convenient method of estimating the amount of uric acid that may be contained in a given specimen. Pavy's test,[1] to which Dr. Da Costa alludes, takes some half-hour of time; so that, unless the urinary peculiarities are visible to the naked eye or the microscope, few men have the time to make the necessary examination. Even if a handy method of quantitative analysis were known, no one has pointed out what amount is necessary to constitute the morbid condition of so-called lithæmia—a very fatal omission when it is remembered that the uric acid secreted daily by healthy individuals varies very greatly in amount. Dr. Roberts[2] observed a difference of nearly one-half on two successive days, on the first day 5.45 grains being separated, on the second 11.7 grains. In three healthy students, living on a similar diet and under similar circumstances, he obtained these figures:

No. 1 (mean of 47 days)	8.051 grains.
No. 2 (mean of 5 days)	3.462 "
No. 3 (mean of 3 days)	6.071 "

Sir William Roberts maintains that uric acid in the urine is the result of the decomposition of the quadri-urates, and that it does not exist in the blood in any other form.

3. *Simple neurasthenia.* By this I mean the set of symptoms which constitute the symptom-group of a true neurasthenia. In a general way, it may be said that the functions of the nervous system are below par. The patient complains of weakness, either mental or physical, or both, and any exertion is apt to be attended by symptoms of irritability of the nervous system. An overworked business man will come complaining of incapacity for his work, and that when he attempts to force himself to perform his professional duties he will have a sensation of cerebral fulness, or sharp pain at the vertex, or an inability to exert those finer mental qualities which he has possessed in the past. The celebrated author, Washington Irving, who wrote one of his later works against time, used to complain that his head

[1] Lancet, 1875.
[2] Urinary and Renal Diseases, Philadelphia, 1872, p. 77.

felt like a sponge squeezed dry at the end of a particularly hard day's labor. Or a woman may be exhausted by household cares, parturition, or the anxieties of a family, and complain of a continual weariness, so that she is obliged to spend most of her time reclining upon a couch. The cerebral form of exhaustion is perhaps least familiar to general practitioners, but those who deal with mental disorders are perfectly acquainted with it, for the constant difficulty after recovery from the curable insanities is that patients are apt to become so much discouraged for a long time with their inability to perform any sustained mental labor, and many ambitious students at our large colleges, especially those who have stood high in their classes, at the same time that they have given much time to athletics, have a degree of cerebral exhaustion which sometimes incapacitates them for years after leaving college, perhaps with honor. Any person whose nervous strength is impaired from any cause whatsoever is, of course, liable to erratic nervous manifestations, simply because of weakness. Every physician is familiar with this fact and has seen it exemplified in the hysterical weeping of strong men in hospital wards, in the nervous tremblings, in the flushing of the face, and in a thousand and one different ways; so that one would expect to find symptoms of nervous irritability even in these cases of pure neurasthenia; and we do find them, but not nearly so often as one would expect or as in the reflex and so-called lithæmic neurasthenias. We should bear in mind, however, that the hysterical, hypochondriacal, or neurotic individual may be afflicted with a genuine neurasthenia, when it will be a very nice question to determine the relative proportion causatively of the predisposing neuroses and the true neurasthenia. Nevertheless, I wish to lay great stress upon the fact that most cases of the true neurasthenia which I am describing are not complicated with true hysteria or hypochondria, although there may be occasional emotional disturbances and considerable anxiety about the condition of health, as is very natural.

Causation. The causes of reflex neurasthenia are to be found in some one of the non-nervous organs, either the kidneys, the naso-pharynx, the eyes, the heart, the liver, the genitalia, and in such vicious habits as masturbation, alcoholism, morphinism, and cocainism. In all cases of neurasthenia, therefore, these organs should be carefully examined. Disease of the kidneys can easily be determined by careful quantitative, qualitative, and microscopic examinations of the urine. In the naso-pharynx the most frequent causes are, in my experience, diseases of the nostrils, such as hypertrophies of the turbinated bones, deflections of the septum, and pathological septa, which interfere with proper respiration. In the eyes errors of refraction and ocular insufficiencies causing exophoria undoubtedly do cause an occasional neurasthenia. Great stress has been laid of late years upon exophoria as an etiological factor, and there has been fierce discussion among oculists about this question, so that for some time I was myself in considerable doubt as to the exact truth of the matter. I have seen a number of cases of neurasthenia which obstinately resisted all forms of treatment until the exophria was removed. Nevertheless, I believe that

the frequency of this form of neurasthenia is much overestimated, and I also believe that even in this type it is very rarely that operation upon the ocular muscles is called for, inasmuch as the use of prisms is generally quite sufficient. The form of neurasthenia caused by ocular insufficiencies bears so close a resemblance to the symptoms of the lithæmic type that the differential diagnosis is often difficult to make. The cardiac disturbances that are most apt to cause neurasthenia are a nervous heart, next valvular troubles, and then a fatty heart. Hepatic disorders are not frequent causes, in my experience, of neurasthenia, contrary to the general belief of the profession; and, as I shall have occasion to explain in a few minutes, the functional hepatic derangement of the so-called lithæmic form is only one of several symptoms, and it is a question as to whether it is a cause or an effect. These same remarks are true as to stomachic disorders. As to the genitalia, derangement of the vagina and uterus and the uterine appendages frequently causes neurasthenia, although, as in the case of the eyes, I think the frequency of this causative factor is much exaggerated. Alcoholism, morphinism, and cocainism are frequent causes.

Pathology. The pathology of lithæmic neurasthenia has been involved in considerable doubt. The theory that has been advanced by Murchison has met with general acceptance and was based upon some undeniable clinical facts—as that in gouty persons uric acid was to be found in the blood, as Garrod had shown, and that uric acid and the urates in seemingly large quantities were also to be found in the urine of individuals of gouty heredity or gouty tendencies—and upon one assumption, which was that therefore these neurasthenics were to be regarded as suffering in a less degree from the same underlying hæmic troubles as were the gouty. Thus supported, the theory was elaborated with a wealth of learning and a charm of style that made it attractive, the more especially as it was so very simple. "When oxidation," says Murchison, "is imperfectly performed in the liver, there is a production of insoluble lithic acids and lithates instead of urea, which is the soluble product resulting from the last stage of oxidation of nitrogenous matter. . . . When more food is taken into the blood than is necessary for the nutrition of the tissues the excess is thrown off in the form of urea, carbonic acid, and water, or in the imperfectly oxidized forms of lithic acid and oxalic acid. Under these circumstances the excess of uric acid is thrown upon the liver and other organs, and one result is that a quantity of albumin, instead of being converted into urea, is discharged by the kidneys in the less oxidized form of lithic acid and its salts. But what in most persons is an occasional result of an extreme cause is in some almost a daily occurrence, either from the food being always excessive in amount, or unduly stimulating, or from some innate defect of power often hereditary in the liver, in virtue of which its healthy functions are liable to be deranged by the most ordinary articles of diet. Most persons appear to have more liver, just as they have more lung, than is absolutely necessary for the due performance of its functions; but in others of gouty appear-

ance the organ in its natural condition seems only just capable of performing its healthy functions under the most favorable circumstances, and functional derangement is at once induced by articles of diet which most persons digest with facility. This functional derangement may manifest itself by various symptoms of indigestion, by disturbances of circulation and of other physiological symptoms, but especially by deposits of lithic acid, lithates, and pigments in the urine." I am by no means a votary of this belief in the dogmatic and limited form in which Murchison has stated it, but I am thoroughly convinced that lithæmic neurasthenia bears some relationship to a condition of mal-excretion, because a large proportion of the cases occur in individuals whose urine will be found to contain undue quantities of the amorphous urates and uric acids and whose stools are defective in quantity and proper bilious coloring; because many of these individuals are of rheumatic diathesis, and the English writers state that abroad these individuals are generally of gouty predisposition, which is by no means generally the case in this country; because many cases can never be successfully treated unless this defect of excretion is remedied partially or entirely; because some simple cases will end in perfect recovery with no other treatment than this; and because the cases often follow an attack of malaria, or occur in malarious individuals at malarious seasons of the year. But Murchison's theory of lithæmia is too radically wanting in positive proof for us to accept it in its nakedness, or to do more than to use the term as a convenient clinical designation for a certain group of symptoms. At the outset it should be understood that the name lithæmia is a misnomer, because it is a pure assumption to say that uric acid is to be found in the blood in this condition, as it has never been demonstrated, although it is an undoubted fact that it is found in the blood of gouty people. The older name of lithuria was undoubtedly a better one, but a still better one would be *lithoidosis,* meaning a lithic-acid-like diathesis, as more comprehensive and less dogmatic and more permissive of future developments. The theory that uric acid is a product of sub-oxidation relatively to urea has been met by the fact that in birds and certain reptiles,[1] representing the maximum and minimum of the oxidative processes in the living series, uric acid is to be constantly found in the urine, taking the place of urea. As a remarkable demonstration that oxidation can be no great factor in the production of the phenomena with which uric acid is associated is the further fact that gout has been found in these two oxidative extremes—birds and reptiles.[2] Megnin and Bertin found numerous urea crystals in the joints of parrots. Andrinaldi saw chalky deposits in the claws of hawks. Pagenstecher, examining an *alligator sclerops* five days after death, found deposits of urea and soda in all the muscles, also free uric acid in the hip-joint, the chemical analysis being made by Prof. Carius. Liebig saw uric-acid crystals in great number in the muscles of another alligator. Foster's conservative dictum upon this subject would seem to be the only one warrant-

[1] Foster: Text-book of Physiology, 1880, p. 452.
[2] Ebstein: Die Natur und Behandlung der Gicht. Wiesbaden, 1882, S. 51.

able: "All the facts known go to show that the appearance of uric acid is the metabolism slightly diverging from that leading to urea;" nor does the assertion that the excessive formation of uric acid is due to functional disease of the liver rise above the dignity of a pure assumption. The source of uric acid is not known. Von Schroeder extirpated the kidneys in fowls and reptiles and ligated the aorta and vena cava in fowls, and still uric acid was found in considerable quantity, thus demonstrating that it was not produced in the kidneys. Meissner regarded the liver as the main formative organ, although he admits that the spleen and the nerve-centres take part in the phenomena; but Meissner's experiments are lacking in the crucial test of the elimination of the liver, and simply demonstrate the presence of large and constant quantities of uric acid in the livers of birds. Ranke maintains that the spleen is the chief producing organ. Robin, Chrzonscensky, and Colasanti believe that uric acid originates in the connective tissue. Haig would have us believe that uric acid is constantly formed in the blood, to be excreted when this becomes alkaline, and to be deposited in the joints and tendons when this is acid, these alkaline and acid alternations occurring daily. But Sir William Roberts, as has been said, seemingly demonstrates that uric acid is due to the decomposition of the quadri-urates in the urine, and is not found in the blood. If any conclusion can be drawn from these differing experiments, it is that the production of uric acid is not monopolized by any one tissue, but that the spleen, the nervous system, the liver, and the connective tissues all have their share in the formation of it. Let this aspect of the matter, however, be decided as it may—let uric acid be formed in one organ or in all organs—the question still confronts us: Is it a cause or an effect? It is a familiar fact that Claude Bernard produced diabetes by irritating the diabetic centre in the fourth ventricle. Hence the possibility of diabetes being of nervous origin is demonstrated, and the clinical features of many cases are such as to render it extreemely probable that they are due to an affection of the nervous centres. Certain it is that in the whole vast range of medicine there is nothing that has been more conclusively demonstrated, clinically and post mortem, than that the nervous system has a powerful control of muscular action. Atrophy of muscle, conversion of muscular tissue into fat, cutaneous eruptions, sloughs, ulcers, tumefactions of joints and serous effusions into and around them, trophic changes in bones leading to atrophy and fractures—all these are phenomena that are now known beyond question to arise from the diseases of the nervous system. It seems absurd to question that the agency which is capable of so radically perverting cellular action is not also capable of effecting so relatively slight a cellular change in some tissue or tissues as to lead to the production of uric acid. *A priori*, then, uric acid in so-called lithæmia may be a product as well as it can be a cause, and it is a matter of great importance that this aspect of the question should never be lost sight of in the therapeutics of lithæmic neurasthenia. Indeed, no one has yet pointed out what the normal amount of uric acid is in the urine,

and, therefore, no one knows what an excess of it is; and it is a common observation to find the nervous symptoms altogether disproportionate to the quantity of uric acid discoverable in the urine, as well as to observe these nervous symptoms obstinately persisting after the excretory organs are acting freely, or to have certain nervous phenomena, notably the vertigo, increased by the efforts made to stimulate excretion. If I may formulate conclusions based upon many years of treatment, the most frequent cause of lithæmic neurasthenia is functional derangement of the gastro-intestinal tracts—in other words, of the stomach and the small and large intestines—and of the associated glands, the liver, the pancreas, the spleen, and the gastro-intestinal glands, so that measures addressed to defects of gastric or intestinal digestion or to increased excretion of the associated glands practically bring the most success. In some rare cases, too, sluggish action of the large intestine is alone at fault.

I have made but two autopsies in cases of neurasthenia, and in neither of these cases was there anything discovered by microscopic examination of the brain, spinal cord, or peripheral nerves, and in neither case was the death due to the neurasthenia—resulting in one from an intercurrent pneumonia, and in the other from trauma. I have, however, known of three cases of death from pure neurasthenia in which no autopsy was made. I have myself made six autopsies in cases that were improperly diagnosed as neurasthenia, and in which the findings were respectively aneurism of the arch of the aorta, nephritis, cerebral hemorrhage in the early stage of general paresis, enormous atrophy of the large intestine and of the spleen, Weil's disease, and fatty heart. I have also made autopsies in cases with hysterical symptoms misinterpreted as neurasthenia, with different findings that are detailed under "Hysteria." Neurasthenia of the simple, the reflex, or the lithæmic form is one of the so-called functional diseases; *i.e.*, the pathological alterations are either cellular or vascular in so slight a degree that their exact nature has not yet been determined by the aids to investigation that are at our disposal in the present state of knowledge. It is idle to speculate as to whether they are vaso-motor or cellular, in the sympathetic nervous system or in the cerebro-spinal, because we have no data upon which to base any intelligent conclusions. The older writers of a quarter of a century ago wrote very glibly about cerebral and spinal hyperæmia and anæmia; but in Chapter VIII., "Hyperæmia and Anæmia of the Brain and Cord," I attempted to demonstrate that these supposititious vascular disturbances belong to the metaphysics of medicine. We should, however, grasp clearly the fact that the nervous system can itself be in an asthenic condition, and that such an asthenic condition can also be produced by reflexes and by vascular disturbances. Just as a nerve or a spinal cord can be fatigued by repeated discharges of electricity through either one of them, or just as we constantly see that the human cerebrum can be fatigued by excessive functionation of it, so can we easily conceive that the whole cerebro-spinal nervous system or the whole sympathetic nervous system, or both, or either,

or parts of either, can become asthenic; but precisely wherein this asthenia consists, what cellular alterations compose it, whether it is the protoplasm of cells or the nucleus or nucleoli that become affected, or whether, indeed, the cellular constituents of the peripheral nerves may become altered—this is all an undetermined field of investigation. I see no reason to doubt that the same cell that can become organically altered can also become what we call functionally altered, so that the pathological alteration which we call general paresis or poliomyelitis or peripheral neuritis may have its functional analogue in cerebral, spinal, or peripheral neurasthenia, with this difference: that the cell which is originally altered in its molecular constitution cannot be made to return to the normal so readily by reflex influences from other organs and from the surrounding outer environment as is the case with the functionally impaired cell. Extremely interesting in this connection is Hodge's demonstration of the difference between the brain-cells of a homing pigeon that had returned from a long flight and those of another that had been kept in a coop.

CAUSATION. Whatever may be the real pathologieal nature of lithæmic neurasthenia acting as a predisposition, it is often provoked by extrinsic causes, such as—

Changes of the weather;
Fatigue, mental or physical;
Special articles of diet;
Certain wines;
Improper clothing;
Malaria.

In my experience most of the relatively acute outbreaks are apt to occur in the spring or early summer, much more frequently, singular to say, than in the autumn. In some there is especially apt to be trouble at the first onset of hot weather. In our middle and northern States the spring is a very uncertain season, the gradual rise of the thermometer being frequently interrupted by quick and violent changes back to a wintry temperature until the latter part of May or early in June, when the heat may suddenly reach 85° to 90° or even more, and then merge into moderate summer. The changes are not apt to be so violent in the autumn, and when they are, the change is from warm weather to cold, which does not have so great an effect upon this form of neurasthenia.

Fatigue, mental or physical, is a very frequent cause, and will often bring back the nervous symptoms.

Many patients of this class have an idiosyncrasy about special articles of diet or certain wines—far less, in my experience, about the former than about the latter. Thus the fact to which Dr. Wm. H. Draper has called attention, that the gouty cannot digest starchy and saccharine matters, is true to a certain extent. Of these individuals some cannot digest sugar, some cannot digest starch, but the most fail to digest more than a moderate quantity of one or the other; and I have never met those deficient in the capacity to digest both starch and sugar. Others again cannot digest meat during hot weather as well as in other portions of the year. Still others again cannot digest

eggs. The sweet wines, champagne, Rhine wine, burgundy, and the malt liquors are generally borne badly.

Improper clothing will, in some nervous systems that have become susceptible, often bring about a return of the nervous symptoms in an incredibly prompt manner, and this applies not only to too much clothing in warm weather and too little in cold weather, but to clothing that is not made to follow the sudden changes that may occur from day to day in our variable climate. I have known an alarming dizziness or a neuralgia to disappear in a few minutes by the substitution of a thin for a thick coat on a hot day, or the reverse on a cold day. The curious thing is that this need for a change of clothing is often not betokened by a subjective sense of heat or cold or any alteration in the bodily temperature, although in some cases patients come to know by experience that the weather is becoming warmer by a feeling of nervousness and chilliness.

Malaria is often ascertained to precede this affection, and the inefficacy of antiperiodics in some cases of so-called latent malaria is often due to the non-recognition of the presence of this lithæmic neurasthenia.

Prognosis. The prognosis of neurasthenia is excellent, with proper treatment. Indeed, the majority of cases themselves tend to more or less imperfect recovery, although there is a handsome proportion in which improvement does not take place without treatment; but I have found that severe nervous disturbance beginning in the late spring or early summer, especially of the lithæmic variety, cannot be overcome before the autumn, and when the tendency to neurasthenia is once established recurrences are frequent, especially in the spring, and it often becomes hereditary.

Diagnosis. In every case of neurasthenia that is brought to us a careful examination should be made of the urine, in order to determine whether there is present the so-called lithæmic urine or whether there are evidences of nephritis. Careful search should also be made for possible reflex causes, such as lesions of the naso-pharynx, marked errors of refraction, exophoria, cardiac disease, hepatic disease, intestinal disease, malaria, marked lesions of the uterus and its appendages in the female, of the bladder and the urethra in the male, or the prostate in the elderly male, and for any peripheral nerve-lesions. Care should be taken, however, not to attach undue importance to any of these causes unless the onset of neurasthenia has been coincident with the beginning of one of these lesions, or unless it has been shortly subsequent to them. In cases where the history is defective as to this chronological sequence, it should always be borne in mind that neurasthenia may coexist with these affections and yet be capable of being relieved without treatment of them—often aggravating them, indeed. The diseases with which neurasthenia may be confounded are:

Hysteria;
Migraine;
Angina pectoris;
Chorea;
General paresis;

Nephritis;
Brain-tumor;
Certain non-insane delusional conditions often associated with hysterical tendencies and morbid fears;
Melancholia;
Certain mild chronic insanities;
Locomotor ataxia;
Myotonia;
Malaria;
Progressive muscular atrophy, especially of the neuritic form;
Traumatic neurasthenia;
Ménière's disease.

In severe cases of hysteria the characteristic limitations of the field of vision, the anæsthesia or the hæmianæsthesia, the convulsions and the emotional conditions, are symptoms of great importance in the diagnosis. In the milder forms of hysteria the diagnosis is often difficult, and it may be impossible to make it without careful observation for a few days or more; but the tendency to exaggeration, the variability of the symptoms, the emotional conditions which have dated back for years before the onset of the neurasthenia, and which are not in proportion to the amount of neurasthenia, the caprice that shows itself in a myriad ways, are symptoms that are in sharp contrast with the steadiness and persistency and unemotionability of the neurasthenic phenomena. As has already been said, however, it should never be forgotten that the neurasthenic may become emotional; but in such cases the emotion is temporary and superadded to the steady and persistent substratum of continuing neurasthenia. In some cases it may happen, too, that neurasthenia and hysteria may be combined, and in such cases the characteristic symptoms of hysteria—the limitation of the field of vision, the anæsthesia, and the hemianæsthesia—will be conjoined in all their kaleidoscopic variety with the obstinate and enduring neurasthenia. The most brutal of all mistakes, however, is the confounding of neurasthenia with hysteria. This latter disease is too often thought to be what any other disease is not—is too often the refuge of a moral cowardice that cannot look a doubt squarely in the face; and how often is this so in lithæmic neurasthenia, with its vertigo, its sense of distention about the head, its tinnitus aurium, its undue susceptibility to odors, its nervousness, its slight insomnia, its peculiar neuralgia or perverted sensations, its vasomotor disturbances, and often its outward appearance of ruddy health? It provokes a smile, too, to see how often the sufferers themselves are deceived, even when they are medical men. An apologetic air or a deprecating remark is the common prelude to the history of the symptoms.

There are certain families in which migraine or sick headache is found in several successive generations, especially in the female members. Not infrequently in these individuals loss of consciousness will take the place of the migraine, so that the two functional affections may be interconvertible; in others, associated with the migraine

there may be symptoms simulating mild angina pectoris; still others may occasionally have marked hysterical symptoms; and in still others nervous symptoms may run the gamut of these and other forms of functional disturbance. All the individuals of this migraine group are possessed of great nervous energy, and some of the finest mental work of the world is done by them; but it would be a great mistake to confound these symptoms with those of neurasthenia, although persons of this class may have the latter malady, notwithstanding that they are no more subject to it than other people.

It is by no means so rare as one would expect that cases of general paresis are diagnosed as neurasthenia, for the early symptoms of this gradual, insidious, remittent, generalized interstitial encephalitis are often so fugitive and changeable that it is scarcely to be wondered at that their true significance should be occasionally overlooked. In general paresis, however, the dementia is a characteristic symptom from the very beginning, coloring all the symptoms, however slight they may be, in a way that can always be detected by the careful examiner or observer; besides which, there are in the early stage, the peculiar tremor of the face, tongue, and extremities, the characteristic difficulties of pronunciation, the pupillary abnormalities, and the history of exacerbations, in which there are marked vasomotor symptoms, and in which the tremor, speech-defects, and the pupillary abnormalities become temporarily increased. In some few cases of general paresis in which the course of the disease is without exacerbations and is persistent, the dementia is usually more marked; but the symptoms we may lose for diagnostic purposes are more than atoned for by the presence of this latter. When general paresis has advanced to the stage of delusions, they are always stupid and illogical, so-called unsystematized delusions, and in some non-typical cases these are present from the outset.

If a case of nephritis be accompanied by œdema, either general or limited to the eyelids and ankles, the diagnosis can usually be made without difficulty; but in certain cases of chronic nephritis, especially of the cirrhotic form, repeated and careful chemical and microscopical examinations of the urine should be made.

Certain non-insane delusional conditions, such as the morbid fears of places and individuals, or the semi-insanity which is known as insanity of doubt, have been classified by certain authors under the heading of neurasthenia, and this is where Beard catalogued them; but if we are to accept the ordinary symptoms of neurasthenia as the type of the disease as I have already given them, these semi-insanities belong rather to the group of mental disease than that of neurasthenia, and I have put them under the head of functional insanities.

In brain-tumor the headache is often violent and persistent, or is at all events something more than a mere sense of fulness. Neuro-retinitis may be present, while I have never seen it in lithæmia, although Da Costa claims that he has; but I think he has mistaken some organic form of cerebral disease for it. Vertigo is seldom so marked, and seldom occurs in such paroxysms; although in certain cases affecting the cerebellum or its peduncles it may be equally

pronounced, but it is then that of a marked backward, or forward, or rotary character. When vomiting is present it is of the sudden, explosive, seemingly causeless cerebral type; and in most cases there will be some evidence of structural lesions of the intracranial contents, such as paralysis of motion or sensation, aphasia, hemianopsia, word-deafness, or mental alterations.

Many cases of simple melancholia are unaccompanied by illusions, hallucinations, or delusions, and simply manifest, especially in the early stage, the melancholic tendencies—which are usually distinguished from hypochondria with ease, because in the latter the patient is querulous, seeking new remedies and new physicians, the disease lasting usually many years; whereas melancholia comes on in a few weeks, the simpler forms have an average duration of about a year (unless treated properly, when they are usually much shorter), and the patient is utterly indifferent about himself. The melancholiac has, moreover, a peculiar *facies*, which I have described in the section upon "Melancholia," of mixed suspicion and suffering, to which may be added, as the result of the impaired cerebral reflex, an expression of bewilderment, which latter mental condition, if not evident at first sight, may be elicited by careful questioning, as the patient will state himself that he has been sometimes confused in his thought, or is unable to grasp quickly the meaning of remarks addressed to him, or to respond to them readily. Besides this, there is in melancholia the post-cervical ache, the obstinate insomnia, and the inherent tendency to suicidal impulses. I have never seen delusions, illusions, hallucinations, dull cerebral reflex, post-cervical ache, or obstinate insomnia in a case of neurasthenia.

In some forms of insanity running a very mild and chronic course it is possible to confound the symptoms with those of neurasthenia, especially when the hallucinations or delusions are concealed, and I am always very suspicious of any patient coming to my office alone and complaining of symptoms of cerebral exhaustion. A careful history of the patient from friends or relatives will, however, make the diagnosis clear, and thus avoid the mistake of diagnosing a case of acute or chronic paranoia or the early stage of a subacute mania as one of neurasthenia.

The early stage of locomotor ataxia is sometimes confounded with neurasthenia. Physicians are not generally aware that the early symptoms of this disease are often of a purely sensory nature, and that the ataxia may belong to the later stage. The lightning- and stabbing-pains, severe, sudden, seldom fixed in any one part of the body; the peculiar pupil described by Argyll-Robertson—contracted, irresponsive to light, only feebly responding to movements of accommodation; the loss of the knee-jerk; the slight vesical troubles; the ataxia when present; and the frequent atrophy of the optic nerve: these early symptoms of locomotor ataxia may be overlooked unless they are specially asked and looked for.

It is by no means unusual in my experience to see cases of progressive muscular atrophy confounded with neurasthenia. Modern observation, more particularly in the last ten years, has done away

with the old doctrine that progressive muscular atrophies commenced in the thenar and hypothenar eminences (see section on "Progressive Muscular Atrophy") As a matter of fact, most of them begin in the muscles of the limbs, the shoulder-girdles, or the trunk, and it is sometimes a very difficult matter to determine as to whether there is an actual atrophy. Thus, when the patient complains of a vague loss of strength and vague aching pains, such as are apt to occur in the neuritic forms of muscular atrophy, it may be very easy to overlook the malady unless the patient were stripped and the different muscles and their outlines and the contours of the different muscular groups were keenly observed.

In traumatic neurasthenia the history of the trauma will be of crucial importance; and then the tendency to emotionalism is greater by far than occurs in ordinary or lithæmic neurasthenia.

Ménière's disease has a predominance of aural symptoms of great variety (see section on "Ménière's Disease"), to which may be added a neurasthenic condition; whereas in neurasthenia the most pronounced aural symptom, often absent, is a *tinnitus aurium*, which is not explained by any lesion of the external, middle, or internal ear.

TREATMENT. The treatment should vary accordingly as the case can be classified under reflex, lithæmic, or simple neurasthenia.

In the reflex form careful attention should be paid to the removal of the reflex irritant so far as that may be possible; but, as I have already said, one should not be too hasty in reaching the conclusion that a coexisting minor trouble of a non-nervous organ is the sole cause of neurasthenia, for it has been my experience that the purely reflex form—that is, neurasthenia entirely due to a reflex irritant—is rare, whilst the majority of these reflex cases are caused by a reflex irritant acting in conjunction with other causes; and it often happens that, on the one hand, removal of some reflex irritant has a slight effect or none upon the neurasthenia, whilst in others the neurasthenia may be completely removed, although the supposed reflex irritant remains. Treatment should be addressed, then, to any marked lesion of the naso-pharynx, of the heart, of the kidneys, of the stomach, of the male or female genitalia, of the eyes, or of the ears. If then the neurasthenia persists, or if the supposed reflex irritants are really slight affections, the neurasthenia should itself be treated.

In the so-called lithæmic variety the treatment has been for over a decade a source of great perplexity to me, and it has only been of late years that I have gradually settled down into a routine which has been justified by the large percentage of success that it has insured. First of all, I divide my cases of lithæmic neurasthenia into two classes: those that are sthenic, and those that are asthenic. In the former I begin by giving them a drachm of Carlsbad salt in a half-tumbler of tepid water every morning before breakfast, and I also then give them a glass of Vichy, three or four times a day, about two hours after meals. I pursue this treatment for three or four days and watch the results. If the patient improves rapidly, I then continue the Vichy, and also give a laxative in such doses

as to cause at least two easy, feculent stools in the twenty-four hours. The best laxative for this purpose is the cascara sagrada, which I order to be taken in sufficient doses to attain the effect I have spoken of, and this is usually done by either the solid extract in 2-grain doses, one to three times daily, or the fluid extract in drachm doses the same number of times. My rule is to have the patient determine whether one or three doses a day are necessary, so that he may commence by taking the first dose at bedtime, and, if that does not give the desired movement, take the second one between breakfast and the mid-day meal, the next between the mid-day and the evening meal, and whatever dose they find necessary they are directed to continue using. Occasionally calomel, 3 to 5 grains at bedtime with 10 to 15 grains of bicarbonate of soda, will assist materially; but I never use calomel continuously, because, for some reason that I have never understood, it is apt to increase the vertigo, the nervousness, and sometimes even to weaken the patient, this latter result being often observed with small doses. If this treatment does not have a happy effect, I then discontinue the alkali altogether, and use nitro-muriatic acid, 20 drops three times a day in a wineglass of water just after meals, taking care at the same time to continue the laxative. Sometimes the acid will do best, sometimes the alkali, although it is generally the latter, and I know of no means of determining beforehand which will be best. If neither the acid nor the alkali, with the laxative, cause improvement, or if they cause only a partial improvement, I then begin to probe the intestinal tract, as it were, and this I do by the use of the digestive substances or the intestinal antiseptics, such as salol and naphthalin. Salol should be given in the form of compressed tablets, tablet triturates, or capsules, in doses of gr. ijss. three times daily, although occasionally gr. v. may be needed at the same intervals. If the patient has difficulty in the digestion of nitrogenous substances, evidenced by flatulence or discomfort immediately after meals, I use pepsin; or pancreatin if the symptoms of indigestion appear an hour or two after meals; or salol or pancreatin if the stools are lumpy, or irregular, or sometimes hard and sometimes loose, or if there is great flatulence. If I can obtain no evidence from the patient's statements or symptoms as to whether the stomach or small or large intestine is affected, I am not by any means debarred from the use of these substances, for experience has shown me in many cases that there may be functional conditions of these various portions of the intestinal tract that are reflex irritants, although they may produce no localized symptoms, so that in such cases I systematically use first pepsin; then, if that does not effect any relief, pancreatin; and then, if that in its turn brings no amelioration of the symptoms, salol or naphthalin. The pepsin should always be of an indubitable make, and should never be given with alcohol, except possibly in the form of some light wine, and in many cases it cannot be given with an acid, contrary to what is often advised by therapeutists. If pancreatin is used, it should also be of an indubitable make, because with both pepsin and pancreatin I have found, as has been shown again and again by experiments, that

many of the preparations on the market are absolutely inert. If neither the pepsin nor the pancreatin have brought any relief, I then use salol in $2\frac{1}{2}$ to 5-grain doses in capsule, tablet triturate, or compressed tablet, three times a day for a few days. If this also proves useless, I use naphthalin, which I administer in a carefully made suppository of cocoa-butter with about three drops of castor oil, having the patient insert one of these morning and evening, great care being taken that the cocoa-butter is fresh and soft, so that it will melt readily at the temperature of the rectum. By the use of the alkali or acid with laxatives, or by the use of one or more of these digestives, I succeed in the majority of the sthenic cases. In these patients I also regulate the diet, and in this I am guided by the patient's statements as to whether they can best digest the meats or the farinaceous substances and the vegetables, so that if I find any difficulty in the digestion of any one of these three classes, I restrict the amount of the offending articles, or prohibit them entirely for a short time. It is a curious fact, though, that most cases of lithæmia in any form will not bear the reduction in diet well except for a short time, and in none of the forms have I ever seen any difficulty of digestion of fatty substances of food, and in many cases not even of fatty medicines, such as cod-liver oil. In addition to these medicinal and dietetic measures I regulate the exercise, but very cautiously at first. In some I have the patient take a walk regularly once or twice a day within bounds far short of any fatigue, or I may have him ride on horseback, or I may send him to the gymnasium to have gentle gymnastic exercise under the care of some medical superintendent, or, if he is not accustomed to walking or riding far enough to be a long time in the open air, I have him take a drive for an hour or more every day; or, if this last is not within his means, I have him wrap himself up as if for a drive, shut the doors of his room, open the windows top and bottom, and sit quietly in a chair for an hour or more. Indeed, I may say that in all these cases of neurasthenia I believe that fresh air is relatively of much more importance than exercise, and exercise is always a failure if it be followed by fatigue. In the asthenic cases of lithæmic neurasthenia, however, my treatment is very different. In them I use the acid or the alkali sparingly for a short time and with caution; I am very careful not to weaken the patient in the slightest degree by a laxative, and I seldom use the pepsin, pancreatin, salol, or naphthalin—for in these cases, *plus* the peculiar symptoms which have been called lithæmic, and overshadowing them, are the signs of true neurasthenia, and the treatment should always be that of true or simple neurasthenia.

In this latter form the restriction of the expenditure of energy, or, in less scientific terms, *rest*, is the keynote of my treatment, and with this I conjoin a superabundance of nourishing food. I restrict the expenditure of energy according to my judgment of the needs of the case. This seems like a very simple statement, but the application of it is often a very difficult matter, calling for much experience and nice powers of clinical perception. In all cases, however, where I

am in doubt as to the amount of rest that should be ordered, I err on the safe side, and prefer to give the patient too much rest rather than too little, inasmuch as hundreds of cases have reluctantly forced upon me the conclusion, against all the prejudices of my earlier medical training, that too much rest can never do harm, while too little is frequently the cause of failure. I cannot emphasize this point too strongly to express my fixed convictions. If the patient is utterly worn out, nerveless, incapable of any usual exertion, I put him or her into bed, if I can. I say, *if I can*, because there is a great difference in this respect between the two sexes, for it may be said in general terms that it is only too easy to get a woman into bed, and only too hard to get her out, whilst it is only too hard to get a man into bed, and only too hard to keep him there; so that it is often useless to make the attempt with a man, whilst this may even be the case with some women. If I can keep a patient in bed for a few days or a week, I then allow him or her to get up at noonday, and stay up until an early hour in the evening, and, as the case progresses, I allow them gradually to sit up to a later hour in the evening, and then gradually to rise at an earlier hour in the morning, although in all these cases I endeavor to keep them in bed at least until noonday for a period of ten or twelve weeks, and, in some extreme cases, even longer. Many active business men in the full flood-tide of their career cannot go to bed, however, and this is sometimes true of women. In such a case I carefully inquire into the necessities of the patient's life and his habits, and exert my ingenuity to cut off as much expenditure of energy as is possible. There are a hundred ways of doing this in almost everyone's life. A man, for example, may be able to cut off just so much expenditure of energy by riding to his place of business and from it, going to bed immediately after his evening meal, and rising late the next morning; coming home for an hour or so before his dinner, if that be in the evening, and lying down on a lounge; and staying in bed from Saturday evening until Monday morning, and on holidays. It will be seen that in all this I have taken no account of exercise. This is another conclusion to which I have been forced reluctantly by years of experience, in the teeth of my earlier medical training. I have seen again and again that exercise is not only not needful in these cases of simple neurasthenia, or in the asthenic forms of lithæmic neurasthenia, but that it is positively harmful, for the brisk walk or drive which may exhilarate the robust individual will depress the neurasthenic and increase his nervous symptoms. Massage is a method of treatment that has come into vogue of late years, with more credit than it deserves in this form of disease, I think. I have also almost entirely discontinued its use in neurasthenia for two reasons: first, because in the lithæmic patient it increases the nervousness and vertigo, and in the cases of simple neurasthenia it depresses whilst it is not at all necessary. In all these cases, however, as I have stated, I am a great stickler for fresh air without fatigue, and this I obtain in the extreme cases by having the room in which the patient lies flooded with air without a draught whilst he is warmly covered; or, in the cases

which can stand the fatigue of it, by a drive in the open air, but this should never exceed an hour or an hour and a half. Unless the case of neurasthenia be extreme, these measures of rest will usually increase the appetite and the digestive powers so that excellent quantities of food will be taken without special direction from the physician. If this does not come about, however, I systematically feed the patient. First, I take care that he has three good meals a day of well-cooked food in great variety, not permitting him to limit himself to any few special articles. Good cooking is of quite as much importance for these people as is good food, although it is not necessary to have all the arts of a *chef* or a *cordon bleu*, but simply that the food shall be properly prepared and properly served. I have again and again seen a patient whose nutrition had been a source of anxiety gradually take large quantities of food that were prepared in the proper manner. After he has been taking the three meals for four or five days, I then order a glass of milk every three or four hours, and this quantity I rapidly increase, so that in the course of a week he shall be taking a quart of milk in the twenty-four hours. At the end of a week or ten days, to the milk and the three meals a day I add the equivalent of a pound of beef in the form of beef-tea or beef-extract. When I use the former, I prepare it by the formula given below,[1] or I advise one of the beef-extracts. It must be borne in mind, however, that in the majority of cases beef-tea or a beef-extract can only be given for a short time, as most stomachs revolt against its prolonged use, and in most of the cases, too, which are not extreme enough to need rest in bed, beef-tea can be taken for only a few days. Although this is the rule, there are nevertheless many exceptions to it, and beef-tea or a beef extract should always be tried when needed. Finally, at about the tenth day I commence the use of a malt-extract, and for this purpose I prefer one of the alcoholic extracts, of which I give 2 ounces with each meal, or I use one of the freshly prepared non-alcoholic extracts. The treatment of these cases of simple neurasthenia, or of the asthenic forms of lithæmic neurasthenia, should be continued for at least twelve weeks, and then the patient should keep up the dietetics and the medicines for such time longer as the physician may deem necessary, but he well only need then the occasional supervision of the latter. The tonics which I have found most useful in the simple and the asthenic forms and in the lithæmic variety are iron and the sulphate of strychnine. The iron should be in the form of the dialysed iron, a drachm to 2 drachms three times a day, or Dree's albuminate of iron, a drachm to 2 drachms three times a day, or the peptonate of iron, in compressed tablet, 3 grains three times daily, or

[1] This formula I owe to Dr. Weir Mitchell's book *On Fat and Blood-making*, and if it serve my reader in as good stead as it has myself for ten years, both will be content. Chop up one pound of raw beef, and place it in a bottle with one pint of water and five drops of strong hydrochloric acid. This mixture stands on ice all night, and in the morning the bottle is set in a pan of water at 110° F., and kept two hours at about this temperature. It is then thrown on to a stout cloth and strained until the mass which remains is nearly dry. The filtrate is given in three portions daily. If the raw taste prove very objectionable, the beef to be used is quickly roasted on one side, and then the process is completed in the manner above described. The soup thus made is for the most part raw, but has also the flavor of cooked meat. It should be seasoned to suit the palate of the patient.

ferratin, gr. iv.–viij., three times daily. The dialysed and albuminate of iron should be administered in a cup of water immediately after meals. The sulphate of strychnine I use in the cases which I am permitting to get up out of the bed, or in very asthenic ones in which the rest can only be kept up for a limited time that is really insufficient, using it in these cases as an invaluable cerebro-spinal and cardiac stimulant. The dose should be $\frac{1}{50}$ to $\frac{1}{25}$ grain three times a day, generally the latter. Occasionally cinchona bark may be employed, preferably in the form of the officinal elixir in half-ounce to ounce doses three times a day, but usually cinchona does not agree with these cases. Sulphate of quinine usually acts as an irritant to the nervous system. Electricity is a very valuable adjuvant in the treatment of lithæmic neurasthenia in both its forms, and of the simple neurasthenia—either by the faradic or galvanic currents. The former may be employed by a modified faradization, obtained by having the patient place the bare feet upon a large electrode, and putting both hands upon another large electrode, turning on a current just gentle enough to be appreciated by the patient, and continuing to increase the amount gently as the patient becomes less perceptible of its effect. Where a patient is confined to the bed, a nurse or attendant may gently pass two small electrodes (Fig. 71, or the roller electrode, Fig. 80) about three inches apart, with a gentle current, all over the body, taking fifteen to thirty minutes for the sitting. This is quite as effective as stripping the patient, putting him or her on a lounge, and going through all the cumbrous procedure of general faradization that is generally recommended in the text-books, besides which it is a saving of time of no mean importance to a general practitioner who may have to treat the patient in his office, or who may not be able to obtain a proper attendant to give electricity to him at his house. Galvanism should be of the spinal cord and of the brain. In the former case, one large electrode (Fig. 68) should be placed at the nape of the neck, the other (same, Fig. 68) on the dorsal spine, and a current of 3 to 5 milliampères should be carefully measured off by means of the rheostat, and continued for from three to five minutes. Cerebral galvanization should be by means of an electrode (Fig. 67) at the nape of the neck, another one on the brow (Fig. 69), and a very moderate current of 1 or 2 milliampères, rarely 3 milliampères, should be very gradually turned on and passed at the first sittings for two minutes, then for from three to five, taking the most extreme care not to move the electrodes or to jar the patient, and then the current should be as carefully turned off. Useful, however, as faradism and galvanism are, there are many cases which can be conducted to a successful issue without them. The static current I have never found of much use except as a light and pleasant stimulant in the stage of convalescence, and then the electric air is the most beneficial.

It will be seen by all this that I am no believer in the heroic methods of treatment which have been so generally advocated in neurasthenia. If I am asked why I should be so dogmatic about the matter, my answer would be simply that my dogmatism has been

forced upon me by a long series of mistakes in my earlier days, and since I have reached my present conclusion I welcome cases of neurasthenia, whereas I formerly dreaded them. Cutting down the diet, violent exercise, heroic doses of purgatives, especially calomel, restriction of food to one particular article or articles, travel, diversion of interest—have again and again failed at my hands. I do not mean, however, to say that the patient should be permanently restricted from healthful outdoor exercise. On the contrary, when health is again established, I always advise that exercise should be systematically begun, taking care not to have any fatigue attend it. Observation has convinced me that even this advice must be cautiously given, as in these cases it is often years before full exercise can be taken with impunity. Even in cases which have been brought to this successful point, and much more so in the ones which have not been fairly restored, there are certain seasons of the year in which the patient should be very careful to avoid any fatigue, and these are usually the spring months in the variable climate of our northern and middle States, and the summer. At these times the patient, who has thought that health had been restored, will be often completely discouraged by a return of the old symptoms, especially in the lithæmic variety. The best means of removing these will be as much restriction of energy for a few days as is in any way possible, with perhaps an easy laxative and the use of an acid or alkali in the lithæmic forms. In these slight relapses, due to the weather, it may often be advantageous also to restrict the food for a day or two, either by cutting off meat or by reducing the quantity of all the food taken; but this restriction should be only temporary, as continuance of it may bring about, in its turn, a return of the old symptoms.

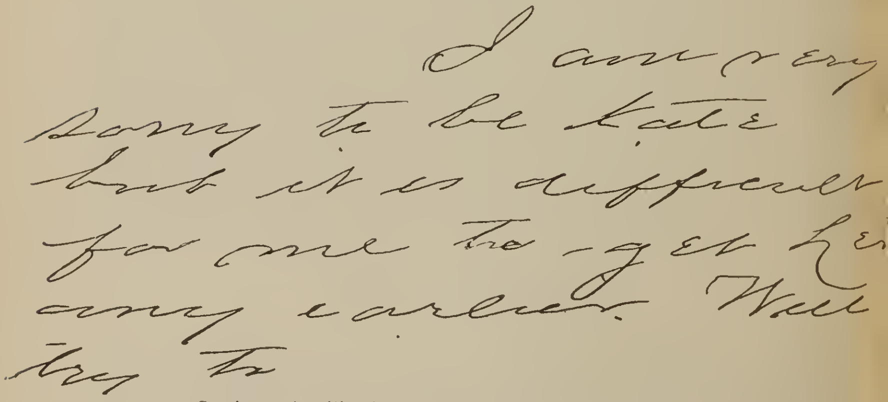
OCCUPATION-NEUROSES.

The term "occupation-neurosis" has been suggested by Gowers to designate certain curious muscular spasms of inco-ordination excited by excessive use of certain groups of muscles in certain occupations. The German term is *Beschäftigungs-Neurosen*. Hammond has coined the phrase, *anapeiratic*, from ἀναπειράω, to do or attempt again; but I think that Gowers' term is a simpler one. These occupation-neuroses consist of writers' cramp, or scriveners' palsy; pianoforte-players' cramp; violin-players' cramp; seamstresses' cramp; telegraphers' cramp; and smiths' cramp. The names indicate sufficiently what they are. In all these the main symptom is muscular spasm, although in certain cases there may be neuralgic symptoms and foci. They are mainly diseases of the active period of adult life. A neurotic predisposition may be traced in some of them. The predisposing causes are sometimes overwork and nervous depression, but the exciting cause is almost invariably actual or relative overstrain of the affected extremity. In some cases the exciting cause may also be a local disease or injury.

The symptoms vary according to the muscles affected. In writers' cramp the muscles of the hand or the forearm are usually affected,

although the whole upper extremity may suffer. According to some authors, this writers' cramp depends largely upon the way in which the pen is held, there being a greater predisposition to it in those who write crabbedly and with the small muscles, using the little finger or the wrist as a support. I have, however, not been able to find that this distinction is of much etiological importance. The patient finds that his hand becomes spastic and tremulous after writing for a time, so that he is unable to co-ordinate in the usual free way. This may vary, of course, in degree. The following specimens of handwriting will show the difference between the first writing and that which is done when the tired feeling come on. In other cases the patient may be absolutely unable to write. The pen cannot be properly held or guided, slipping away with involuntary, rigid, tremulous movements that are very characteristic. If the patient is stripped and the whole upper extremity is examined, it will be found that this is generally weak, and that when the physician's hand is grasped with the utmost strength of which the patient is

FIG. 162.

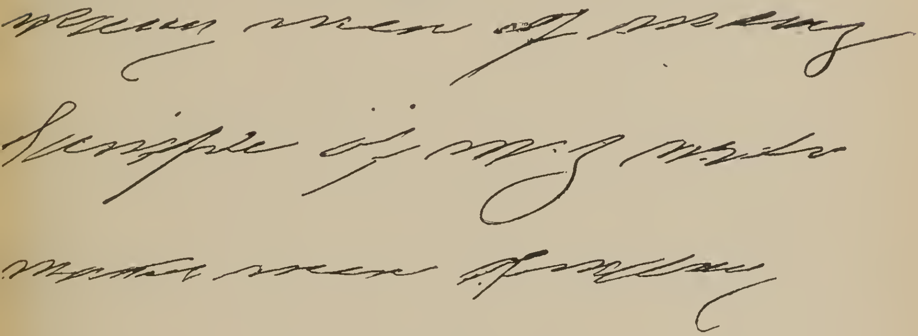


Specimen of writing in writers' cramp when the hand was not tired.

capable there is a singular consentaneous movement of the muscles of the upper arm and shoulder that is relatively far too marked for the amount of strength that is manifested in the hand-grasp. If the sound arm is observed by grasping the hand in a similar manner, the difference between the muscles of the affected and the unaffected extremity can be clearly seen. The muscular spasm and sensory symptoms may extend to the muscles of the neck, and even to the other extremity. In some cases other co-ordinate acts than those of writing can be done perfectly well; but in most there is some slight awkwardness about any acts of the hand requiring continued co-ordination. In pianoforte-players' cramp one finger or a thumb may fail to act in co-ordination with the rest of the fingers, so that it may remain extended or not move in harmony. In violin-players' cramp the same affection may be met with in the finger of either hand, that which moves the strings or bows, and sometimes in both.

In seamstresses' cramp it is the thimble-finger of the right hand that is most often affected. In telegraphers' cramp it is the one finger accustomed to tapping the instrument that is most affected, and if another finger is substituted for this, it may also become implicated. In the cramp of blacksmiths or metal-workers various muscles may be affected, according to those most used. The treatment of any disease should be based to some extent, at least, upon our knowledge of the pathology; and, as I have already said, we have no knowledge of the *modus operandi* of the different forms of muscular cramp and inco-ordination constituting occupation-neuroses, except that they are caused in certain individuals by actual or relative overuse of the affected muscles. In writers' cramp Gowers states that the majority of cases occur in those who use the fingers alone in writing, and who do not consentaneously use the forearm and the upper arm, and he therefore recommends that every patient afflicted with this malady

FIG. 163.



Specimen when the hand was tired.

should lay the arm and forearm upon the table and write with the wrist- and forearm-muscles mainly, using the fingers only as necessary guides. My experience, however, has not confirmed Gowers' observations, for I have not been able to ascertain that the malady is prone to occur in those who use the finger-muscles in writing, nor have I found that using the arm and forearm, as well as the wrist and fingers, will be of more than temporary use in those who already have the disease. Indeed, I have never been able to cure a case, although a number have been reported as cured by other observers. I have in several cases made persistent use of galvanism, faradism, and the bromides, but have never observed more than temporary benefit. A teacher of caligraphy, named Wolfe, is said to have cured a number of cases, and to have given great relief to several others. He entirely failed in two cases of my own. His method consisted of massage and gymnastic exercises of the finer muscles affected. In two cases I have seen great relief obtained by entire disuse of the affected muscles so far as writing was concerned, but in a number of other cases abstention for several years has proved valueless. I do not believe, therefore, that a

patient afflicted with writers' cramp can be treated successfully. This, of course, will often prove a great hardship to those who are dependent for their living upon the use of the pen, and in a great many instances the patient can do his writing quite as well with a typewriter or by means of a phonograph, or can learn to write with the left hand, although this may also in course of time become similarly affected. The sensory symptoms which are sometimes observed in writers' cramp can generally be greatly improved by the use of galvanism, one electrode (Fig. 68) being applied to the cervical spine, and another roller electrode (Fig. 80) being gently passed over both surfaces of the hand and forearm with a current of 5 to 10 milliampères, in sittings of five to fifteen minutes every second day for a period of six to eight weeks. Bernheim claims to have cured several cases by means of hypnotism, and suggests that this is the *modus operandi* of the cures that have been effected by Wolfe. I have had no experience with hypnotism in treating these affections, but I have seen one apprehensive girl cured of a piano-player's cramp by placebos.

CHAPTER X.

NERVOUS DISEASES OF PROBABLY MICROBIC ORIGIN.

TETANUS.

DEFINITION. Persistent tonic spasm with violent exacerbations of the muscles of the jaw and trunk, and secondarily those of the limbs.

ETIOLOGY. The etiological factors of tetanus are—

Sex;
Age;
Race;
Locality;
Injuries;
Dampness and cold or extremes of temperature;
The puerperal condition.

It occurs much more frequently in males than in females, the proportion of the former being about six to one of the latter in the traumatic cases, somewhat less in the idiopathic.

Most cases occur from ten to forty years of age, and it is seldom seen under five, although it is occasionally met with in old age. Tetanus of the newborn is very common in some tropical countries, especially among the colored races, but it is rare in temperate climates.

The negro and the East and West Indian races are more prone to the disease than the Anglo-Saxon or the Latin peoples, even where both are exposed to the same climate.

Traumatism plays a considerable part in the causation of tetanus, and it is said to be most common after punctured, contused, and lacerated wounds and extensive burns, whilst it is uncommon after incised wounds or surgical operations. Upon the southern shore of Long Island, to which allusion is made in the following section of this chapter, it is supposed to be very common after gunshot-wounds or mere pricks, and especially after lacerations inflicted by oyster-shells. The interval between the injury and the first symptoms of tetanus is usually from four to sixteen days, but it may set in earlier or later—in one case a negro cutting his hand with a piece of porcelain being dead in fifteen minutes; on the other hand, it may not commence till the fourth or fifth week afterward. Injuries to small nerves are supposed to give rise to it more frequently than injuries to nerve-trunks, and it is said that it has sometimes followed such traumatisms as flogging and falls.

Beard called attention in 1877 to the frequency of tetanus upon Long Island, which, as most Americans know, is an island about 120

miles long, running southeastwardly along the shore of the Atlantic Ocean, and forming by its western end a part of New York harbor. He illustrates this frequency by showing that whilst in New York City tetanus occurred once in about 30,000 wounds of all sorts, it occurred in this district of Long Island once in about every 200 wounds, or above 150 times as frequently. The inhabitants were prone to attribute it to the use of fish as manure. Of late years, however, indeed for some time before Beard's paper, it would seem to have been dying out. Beard also states that tetanus is more frequent in Bombay, India, than in any other part of the world, inasmuch as in the Jejeebhoy Hospital during seven years, from 1845 to 1851, there were 195 cases of tetanus admitted, and 70 or 80 more were attacked after admission, making 260 cases in all, this being probably, as the reporting surgeon remarks, the largest number of cases treated in any hospital in the world. The influence of different localities may be gathered from the following table taken from Beard's article :

Locality and date.		Total deaths.	Deaths from tetanus.	Proportion.
Bombay, India	1851–1853	42,651	912	1 : 46
New Orleans, La. (whites and blacks)	1847–1873	165,435	1727	1 : 95.7
Charleston, S. C. (blacks)	1846–1865	9,020	93	1 : 97
New Orleans, La.	1849–1850	3.336	34	1 : 99.4
Charleston, S. C. (whites and blacks)	1846–1865	17,150	132	1 : 130
New Orleans, La. (whites)	1849–1850	14,306	96	1 : 149
Nashville, Tenn. (whites, blacks and colored)	1838–1848	....	...	1 : 80
Charleston, S. C. (whites)	1846–1865	8,880	39	1 : 207.1
Augusta, Ga. (whites, blacks and colored)	1845–1866	10,884	34	1 : 320
Augusta. Ga. (blacks and colored)	1845–1866	4,240	19	1 : 228
Savannah, Ga.	1804–1853	14,332	58	1 : 229 8
Philadelphia, Pa.	1807–1827	53,004	125	1 : 424
Augusta, Ga. (whites)	1845–1866	6,645	15	1 : 443
Philadelphia, Pa.	1827–1830	16,579	29	1 : 573
New York	1819–1834	83,783	112	1 : 748
Philadelphia, Pa	1830–1840	49,686	51	1 : 974
Massachusetts	5 years.	104,823	75	1 : 1,907
London, Eng.	1850–3–4	224,515	73	1 : 3,075
Ireland	1831–1851	1,187.374	238	1 : 4,987

Exposure to dampness and cold may be a cause of tetanus. Napoleon's surgeon, Larrey, observed both in Egypt and Germany that tetanus did not often follow wounds unless there were extremes of temperature, and that after the battle of Aboukir ten of the wounded, on being transported to the hospital of Alexandria, were exposed to the cool night air and died of tetanus. François relates that on the frigate "Amazon," before Charleston during the American war, most of those wounded by firearms were attacked by tetanus when wet and stormy weather had succeeded a dry spell. After the battles of Bautzen and Dresden the wounded were exposed on very cold nights, and many cases of tetanus occurred. Baudens relates that while in Africa forty men slightly wounded were placed during the prevalence of a northeast wind in December on the exposed north side of a gallery, and that out of these forty fifteen quickly developed tetanus and twelve died, while the remainder were taken to a more

sheltered position and were not attacked. Kane lost two men and many dogs by tetanus in his North Pole expedition.

The puerperal condition is a frequent cause of tetanus in hot countries, but it is a rare one in temperate climates. Of 50 cases collected by Gowers occurring in temperate climates, it occurred in 18 after abortion and in 32 after labor at or near full term—in most of them there having been severe hemorrhage; in 7 the vagina was plugged, the interval between the abortion and the symptoms varying from five to thirteen days. Of the cases after labor, in all but 3 there was an abnormal condition during or after parturition, the most frequent lesion being adhesion of the placenta, which existed in nearly half the cases; metritis occurring in 2, in association with pharyngeal diphtheria in 1, the forceps having been used in only 1 case, in 1 there having been placenta prævia.

PATHOLOGY. Nicolaier has found that injection of the earth of urban streets into rabbits, mice, and guinea-pigs will often produce symptoms resembling tetanus. As Carle and Ratone have produced a similar malady in the rabbit by the inoculation of the products of tetanus in the human being, it has been deemed probable that the same parasite is the cause in both cases. Rosenbach has been able, he claims, to isolate the same microbe from individuals dying of traumatic tetanus. In inoculating the powder of hay into guinea-pigs, Rietsch has produced tetanus in animals; and the pus formed at the point of inoculation has served to inoculate other guinea-pigs, which died with the same symptoms as the first. The pus of the second series of guinea-pigs has been cultivated in serum, and has displayed the bacillus described by Nicolaier. One of these cultures was hypodermically injected into an ass, which presented, at the end of fifteen days, all the typical symptoms of tetanus in animals, namely: trismus, opisthotonus, contracture of all the muscles of the face, of the dorsal and pectoral muscles, and progressive rigidity of the hind limbs, death supervening upon the twenty-fourth day. The pus and the walls of the abscess from this ass were in their turn injected into rabbits, causing the usual tetanus. Rosenbach has never been able to obtain the pure culture of Nicolaier's bacillus. He believes that this bacillus, which is localized in the wound, acts by secreting a poison whose absorption produces tetanus. From impure cultures of this bacillus Brieger has obtained tetanine, $C_{13}H_{30}Az_2O_4$, producing tetanic symptoms, notably tonic convulsions and opisthotonus. He has also obtained other products called tetanotoxine, $C_5H_{11}Az$, spasmotoxine, and diamine, which all produce cramps and convulsions. Beumer witnessed the death by tetanus of a man of thirty-one, who had wounded himself in the finger with a splinter of wood, and also the death of an infant, who, walking barefooted, wounded himself on the toe with a small pebble; and the particles of earth covering these two objects produced tetanus and cultures of Nicolaier's bacillus. Beumer has also seen in newborn children, who died from trismus at the time of cicatrization of the umbilicus, the umbilical wound and the neighboring tissue possessed of tetanic properties for animals, and giving virulent cultures. Bonardi has

found in the pus from a tetanic wound various microbes, such as abundant cocci resembling the *staphylococcus pyogenes aureus*, *bacterium termo*, the *bacillus pyogenes fœtidus*, and finally an unnamed bacillus of some length, possessing a spore at one of its extremities. He was not, however, able to obtain pure cultures of these different microbes. Other authors, however, have obtained such pure cultures of the long bacillus with a spore, and this is regarded by Nicolaier and Rosenbach as the true tetanic bacillus. Some doubt, however, has been cast upon the matter, especially by Kitasato. In all cases of tetanus observed by Cornil and Babes the microbe of Nicolaier was not found. In another case of tetanus originating in a double tuberculous otitis it was also absent. In the laboratory of Flügge, where Nicolaier did his work, it was ascertained that, in a case of tetanus, caused by a wound with a splinter of wood, the wood produced tetanus in animals, but neither the wood nor the organs examined after death, nor the wound itself, contained Nicolaier's bacillus. Flügge has also reported two observations of the same kind. Chantemesse and Vidal, working in the laboratory of Cornil and Babes, have been able to isolate and to cultivate in all its purity the bacillus of Nicolaier from the wounds of tetanic men and animals. These observers have also ascertained that in a ward where tetanus had often been small quantities of dust taken from the walls, the furniture, and the iron casing were all inert, except one particular dust containing earth taken from the grooves of a window-grating. The inoculations made from the organs of a woman dying with tetanus were inert when injected into animals, with the exception of that taken from the mucous membrane of the uterus, and this produced marked tetanic symptoms. Chantemesse and Vidal have demonstrated the marked diminution of virulence in tetanic agents under the influence of air, light, and warmth; for the earth which they took from the window-sill invariably produced tetanus if inoculated immediately after being found, but could be rendered inert by being pulverized on a sheet of paper and left for twenty-four hours in the atmosphere of the laboratory.

Clinical Symptoms. There are seldom prodromal symptoms, and when they are present they usually consist of vague pains in the head. The first real symptom that is noticed is generally a sense of stiffness or rigidity in moving the head or neck or in swallowing. This increases until it becomes difficult to separate the jaws, and there is what is known as trismus, due to rigidity of the masseters. To this succeeds increasing stiffness of the neck, and the head is bent backward. The spinal muscles next become affected, and the vertebral column is bent backward, inducing the condition of opisthotonus. The extremities are not affected to the same extent, although the legs may become stiff and extended. The facial muscles are last affected, and the angles of the mouth are drawn outward and downward, and the upper lip is pressed against the teeth, causing the so-called *risus sardonicus*. The muscles are not at first painful, but there soon supervene paroxysms of pain and rigidity lasting several seconds, during which muscular spasm may

be present to the most extreme degree of opisthotonus, whilst the muscles of the face and abdomen are also involved. Exceptionally, however, the muscles of a limb or of the throat may also become involved. In some cases the rigidity in the abdominal muscles and the flexors of the spine may bend the trunk forward and cause emprosthotonus, or there may be a slight lateral flexion, pleurothotonous, or the trunk and neck become rigid, straight up and down, producing orthotonus. These varieties are, however, exceptional. Usually the muscles of respiration do not participate in the tonic spasm, and when they do there is great danger of death. The spasms are usually intermittent, and they are absent in sleep. Consciousness is never affected at any time throughout the course of the disease. The pulse becomes more frequent and small. The temperature may be normal, or may rise 2° to 5°, and there may be an evening rise or a continuous elevation, the latter without evening remission, although there may be an increase of the bodily heat at each paroxysm and a moderate fall in the intervals; or there may be irregularities of temperature that bear no relation to the symptoms. In some rare cases the temperature may rise as high as 108°, 110°, or even 114°. The urine is scanty and high-colored, although the nitrogenous matter is not increased in quantity, contrary to what is observed in the urine of most fevers. Micturition is usually not affected, except slightly during the paroxysms, but occasionally there may be slight retention. Moderate constipation generally prevails.

When cases do not terminate fatally they gradually diminish and pass off in the course of a few weeks.

Prognosis. The prognosis of tetanus is extremely unfavorable, the mortality varying from 70 to 95 per cent., and in the mortality of newborn children it is about 90 per cent. In idiopathic cases mortality is undoubtedly less than in the traumatic cases, but I have found it impossible to make an average, because, in looking over the literature on the subject, in so many cases that have recovered the diagnosis has been a matter of doubt. In 150 cases that I have seen myself death has occurred in 140.

Diagnosis. This can be understood by reading the section upon this branch of the subject in the following chapter upon "Tetany," which is the only disease that is likely to be confounded with it.

Treatment. In all the cases of tetanus that I have seen common sense would dictate that, in view of the enormously increased irritability of skin and muscle, the most sedulous attention should be paid to keeping the patient absolutely quiet and secluded. The room should be darkened, the attendants should move lightly and gently, their dress should create no rustling noise, and they should wear soft shoes or slippers, whilst all draughts should be carefully excluded, even to the motion of air that may be created by the opening of a door, at the same time that sounds should be deadened in every way that human ingenuity can suggest. Liquid nourishment alone should be given, best by means of a tube through the mouth or the nose; but if this produces spasm, the nourishment should be given by the rectum. Milk, preparations of beef-tea, beef-peptonoids, koumyss,

and alcoholic stimulants should be used. In the traumatic cases any irritable wound should be carefully attended to, and any irritable cicatrix should be excised. I can see no use, however, in excising a wound or a cicatrix unless it is irritable, because this only produces a fresh wound or a fresh cicatrix. The drugs that should be used are chloroform, nitrite of amyl, bromide of potash, chloral hydrate, belladonna, atropine, cannabis indica, aconite, Calabar bean, and curara.

The most efficient treatment I believe to be that of a combination of bromide of potash, chloral hydrate, and opium. Of the former, the commencing dose should be 3 drachms, and of the chloral 1 drachm; and these should be used every two or three hours, and their effect observed. Should this not be favorable, the dose of bromide should be gradually increased to as much as an ounce, and the chloral hydrate to as much as 7 drachms, but these extreme doses should never be given except with the utmost caution. If moderate doses of bromide and chloral do not control the symptoms, it will always be well to try the effect of simultaneous employment of opium in the form of a hypodermic injection of $\frac{1}{8}$ grain sulphate of morphine, and this must be used according to the judgment of the attending physician. Care should always be taken that the morphine has been obtained from some perfectly reliable manufacturer, because the morphine of the shops is too often entirely unreliable, either for the reason that it has been made from cheaper grades of opium or that it has become stale. If Calabar bean is used, the dose hypodermically should be $\frac{1}{2}$ to $1\frac{1}{2}$ grains, or by the mouth 1 grain to 3, and much larger doses have been given, and in none of the cases does it produce the usual effect upon the pupil. Atropine given at the same time is said to counteract the constitutional effects of the Calabar bean. Curara has been given in doses running from $\frac{1}{100}$ to $\frac{1}{2}$ grain every hour, but it is a very dangerous drug to use. Inhalation of chloroform is one of the most effective measures, but I have usually preferred to use it only to control the more violent paroxysms, trusting to the bromide, chloral, and morphine for the continuous treatment of the disease. Whatever drug be used should be continuously administered even after the patient greatly improves.

Simmons suggests that the wound be thoroughly cleansed with a gold needle, that this be attached to a hypodermic syringe, a 4 per cent. solution of cocaine sprayed over the wound, then a solution of nitrate of silver (gr. v., aquæ ℥ss.), or an alcoholic solution of corrosive sublimate and carbolic acid (hydrarg. bichlor. gr. ij., acid. carbol. gr. xv., alcohol ℥ss.).

Artificial respiration should always be employed when seeming death has occurred during a paroxysm, because it is possible by this means to reanimate the patient again. Such far-fetched operations as tying the vertebral arteries or stretching a nerve have not been warranted by the results. Much can be done in the way of tetanus of newborn children, however, in the way of antisepsis, and this should also be rigidly insisted upon in the treatment of all wounds in localities where the disease is prevalent. Assistants, nurses, and

physicians should be scrupulously clean, washing their hands in a solution of bichloride of mercury, 1 to 500; and the wound should be antiseptically dressed. The use of serum from immunized animals seemed at first of great promise, but it has not proven successful; and, moreover, there is great difficulty in obtaining it promptly in case of need.

TETANY.

Synonym: Tetanilla.

Definition. Tetany consists of tonic, bilateral, intercurrent, usually painful spasms of certain muscular groups, without loss of consciousness.

History. This affection was first described by Sternheim, a practising physician of Altona, in 1830, under the heading of "Two Rare Forms of Rheumatism." In 1831 Dantz described the same affection as *tetanus intermittens*, having no knowledge of the previous article. As usual, the older authors seem to have had some glimmerings of the malady, from Hippocrates down to such writers in the seventeenth century as Imbert-Gourbeyre, Delamotte, Delaroche, Lenoble, Tissot, Ramel, Müller, and Wolf. In 1832 Tonnelé made publicly known the results of the first autopsy. Courvisart first suggested the name of tetany, and since then the literature upon the subject has been abundant. It is a very rare disease in America, and no death from it has ever been recorded by the Health Board of the city of New York.

Clinical History. Tetany consists of tonic intermittent spasms of the extremities without loss of consciousness. These spasms are almost always symmetrical and generally affect the hands first and afterward the feet, generally with flexor spasms. The hands are thrown into a position such as they assume in writing, or such as is taken by the hand of a midwife; but this attitude of the hands is not constant, and they may sometimes be closed like a fist, and occasionally only individual fingers may be affected. In the feet the toes are drawn downward and the instep up, as in equinus. Flexions of the elbow are not frequent, and only occasionally is there adduction of the shoulder-joints. In the knee-joints there may be a contracture of the quadriceps and consequent extension, and very seldom, if ever, flexion in the knee-joint. The contracture may be slight, or so pronounced that the physician cannot overcome it, and usually in these cases of excessive contraction the muscles can neither be extended nor flexed. The jaw-muscles are also generally affected, so that there is trismus, or lock-jaw. When the facial muscles are affected, the *risus sardonicus* is present, and this has been seen on one side alone. In the severe cases the muscles of the neck and chest and even of the trunk are affected, so that opisthotonus may supervene. The tongue is seldom affected, or those of the pharynx and larynx. The eyelids are more often involved, and occasionally the ocular muscles themselves, producing strabismus and diplopia. Whilst the rule is that the spasms are bilateral, there are occasional exceptions, and in several instances the hand alone has been affected, or the upper and

lower extremity on one side. The duration of the spasms varies from a few minutes to a half-hour or two hours, rarely several hours, and in some very rare cases they have been known to last several days. These spasms may be induced by mental excitement and nervousness. In some rare instances so-called intentional spasms have been seen; *i. e.*, a voluntary movement being performed, the muscles entering into this movement become affected by spasm. With these spasms goes usually a certain amount of paresis of the affected extremities, which may last for months after the attacks, and in some instances, when the lower extremities are implicated, the gait is tottering and uncertain. The intervals between the spasms vary in length from hours to days, but the spasms themselves seem to occur with a certain periodicity, especially in the afternoons and at night.

Trousseau called attention to a phenomenon in tetany to which his name has been given, so that it is called by the European observers the *Trousseau phenomenon.* It consists of the fact that pressure upon the affected extremities at certain points will produce the peculiar spasm. The best place in which to do this is the bicipital sulcus or the crural sulcus, but this pressure must be exerted from half a minute to four or five minutes. In certain exceptional cases pressure upon the bones of the hand will suffice, or upon painful vertebral prominences, or pricking with a pin, or sprinkling with cold water. It is not, however, a constant phenomenon, but is of great diagnostic importance when present. It is evidently due to irritation of the nerve-trunks or of the muscular nerve-fibres, and not to any compression of the capillaries or arteries, as Trousseau at first supposed, because experiments have shown that pressure upon the arteries was not sufficient. Chvostek called attention to the fact that the mechanical excitability of the nerves was greatly increased in tetany, especially in the face, and this phenomenon has been called the *Chvostek symptom.* It is evoked by stroking and pinching the muscle with the finger, or in lesser cases percussing the muscle with a percussion-hammer of rubber, when instantaneous contraction of the neighboring muscles will be observed. But this symptom has been observed in other morbid conditions of the nervous system, such as hypnotism, the later stages of facial paralysis, and certain forms of pellagra, and also in healthy young adults, occasionally in healthy individuals of more advanced age. The electrical excitability is almost always increased.

As a rule, the sensorium is unaffected, but it may happen that there is some confusion, delirium, melancholoid states, or confusional insanity. The temperature is subnormal generally, but it may be above the normal or even high. Pulse and respiration are affected, usually in proportion to the severity of the attack, somewhat quickened in the lighter attacks, or very much increased in the severe ones. In almost every case there are increased respiration, redness of the skin of the extremities, especially in the region of the joints, slight degrees of œdema, and there sometimes is even a slightly bloated appearance of the face, occasionally a herpes, urticaria, alterations of the hair, nails, and even, but rarely, muscular atrophy. The knee-jerks may

be normal, subnormal, or exaggerated, and are of no diagnostic value whatever. Epileptic attacks frequently occur with tetany.

Etiology. The causes of tetany are—

Certain seasons;
Certain occupations;
Certain stomachic and intestinal affections;
Certain acute infectious diseases;
Toxic agents;
Parturition and its sequelæ;
Extirpation of the thyroid gland.

The disease is most frequent in infancy and the second decade of life. Heredity is but very rarely an important etiological factor.

The disease is very much more apt to occur in the months of February, March, April, and May than in the other months, and Frankl-Hochwart, analyzing cases occurring among 200,000 patients admitted to the hospitals of Vienna for nine years, from 1880 to 1889, found that the increase in cases generally occurred in January, rose rapidly during February, attained the maximum in March, decreased through April, and in May sank below the point from which it had started.

It would seem that certain occupations are particularly prone to this disease. Thus, shoemakers are the most prone to it; next in frequency, tailors, and then, far less frequently, joiners, turners, locksmiths; whilst saddlers, hostlers, day laborers, wagoners, bookbinders, watchmakers, butchers, coachmen, and masons are singularly free from it.

It may occur after diarrhœa, dyspepsia, worms, constipation, typhus, cholera, measles, scarlatina, and many infectious diseases. It has also been observed in conjunction with ergotism, alcoholism, chloroform, and uræmia, and in pregnant, parturient, and nursing women, as well as from extirpation of the thyroid gland, which procedure probably induces it, as it does myxœdema, by the poisoning of the mucin, since two German observers, Wagner and Hammerschlag, have induced tetany in cats by the injection of mucin.

Pathology. The pathology of tetany is unknown, although it is probably due to some microbe.

Duration and Prognosis. Tetany may be chronic, the attacks recurring at intervals for years, and Hoffman speaks of one such case which had lasted over twenty-one years; but this is rare, as is also a fatal result in the disease. The duration is usually a short one, but the tendency to relapses is often considerable. The duration of the first attacks is usually four to fourteen days. The form which occurs with dilatation of the stomach usually ends fatally, but most of the other cases recover, unless they are mere symptoms accompanying some disease that is in itself fatal. It may occur sporadically, in epidemics, or isolatedly.

Diagnosis. The only disease with which there may be any danger of confusing tetany is tetanus, but in the latter disease the muscular cramps begin in the neck and the muscles of mastication, whilst the hands are not usually affected at this time, and there is an enormous

general reflex excitability; besides, there are none of the facial phenomena or the increase of galvanic excitability, nor can the cramps be evoked by pressure upon certain points (Trousseau's symptom). It is claimed that tetany can exist without cramps, in which case the diagnosis can be made by the increased mechanical irritability of nerves and muscles, the increased electrical irritability, and the presence of the characteristic pains. Hysterical contracture may resemble tetany, but it is generally unilateral, and the differential diagnosis can be easily made by the symptoms.

Treatment. If a cause can be found, it should be removed, as in the case of tetany occurring with dyspepsia or constipation.

Bromide of potash is very useful in overcoming the spasms; from 15 to 20 grains should be administered three times a day, or every four or five hours. In a less degree chloral, morphine, Indian hemp, chloroform, cold to the extremities, and ice to the spine have had an excellent effect. Electrical treatment is of doubtful value. Gowers speaks of digitalis as a most useful remedy for nocturnal tetany, a dose being given at bedtime. In all cases tonics, nourishment, and supporting treatment should be also made use of in accordance with the need of the individual case.

HYDROPHOBIA, or RABIES.

At the very outset of this question some details of nomenclature should be briefly considered. By usage the term rabies is given to the disease in the animal, while the malady in man is termed hydrophobia. This was so stated in Fleming's book in 1872, and has since been adopted by the standard dictionaries and writers. I see no reason, however, why it should not be called rabies in the human subject. The disease was termed *lyssa* by the Greeks, meaning a worm, a term given to the frænum of a dog's tongue, which was supposed in the early days to be a worm, and was extirpated as such. This superstition lingered until the early part of this century, when a Russian physician by the name of Marochetti claimed that in all cases of hydrophobia pustules appeared under the tongue at a certain time of the incubation of the disease. The German writers still retain this name of lyssa. The spread of an infectious disease among animals is called an epizooty (from *ἐπί*, upon, and *ζῶον*, an animal), just as the spread of an infectious disease in the human being is called an epidemic (from *ἐπί*, upon, and *δῆμος*, the people).

At the very threshold of this subject we are confronted by the question as to whether there is such a thing as rabies or hydrophobia. There has been much and bitter discussion upon this point during the last few years. Those who would answer this question in the affirmative allege as proof the fixed belief of ages and the many epizooties of which we have historical record. The very earliest writers speak of rabid dogs, and they were known as far back as the days of the Asclepiadæ, the descendants of the god of medicine, Æsculapius; while there are very few parts of the world, whether in the tropics or the arctics, in which an outbreak of it has not been

described at some time or other. At times it has raged with great fury, as in Northern Germany from 1851 to 1856, when at Hamburg alone 600 cases were observed among dogs, 267 occurring in the year 1851. In England, in 1856, it raged severely among dogs and in a herd of deer, destroying nearly one hundred of the latter. In Würtemberg, according to the veterinary professor, Hering, from January 1, 1840, until the end of February, 1842, it affected 230 dogs and 21 bitches. In 1866 there were thirty-six deaths from hydrophobia reported by the Registrar General of Great Britain. It has never been rife in this country, although local epizooties of it have been reported from time to time. It is stated that it raged two years in a herd of deer belonging to the Marquis of Ipswich. In almost all of these epizooties human beings have been bitten by the rabid animal and have died, although it has never spread from one human being to another, as it has done in animals. It would seem, therefore, to be conclusively proven that there is a disease which can rapidly exterminate a kennel of dogs, a flock of sheep, a drove of hogs, or a herd of deer, and which can render the timid skunk, badger, and fox ferocious and dangerous and make the wolf a beast of prey as fierce as a royal Bengal tiger. It would also appear to be conclusively proven that many people have died after being bitten by these rabid animals. But there has sprung up in this country, in the last few years, a small number of very decided opponents of these claims, such as Spitzka, Brill, Dulles, Stockwell, and others; and, unless I am very much mistaken, they have voiced to some extent the feeling of skepticism that prevails among American physicians. In the first place, they say, many mistakes are made in diagnosis, so that animals are said to have rabies when in reality they have some one of many disorders that may cause similar symptoms, such as epilepsy, angina or sore-throat, gastritis and enteritis, distemper, foreign bodies in the mouth and throat, tetanus, worms in the frontal sinuses or in the small intestine, and ulceration of the internal ear. In certain countries, extending from the sources of the Vistula to the Carpathian Mountains, there is endemic a disease known as the "plica Polonica," attacking mankind, horses, cows, sheep, wolves, foxes, and dogs, in the latter closely resembling rabies, although the bite of these animals is not followed by hydrophobia. Spitzka has introduced into the skull-cavities of dogs such non-rabic material as the spinal cord of a healthy calf, common yellow soap and stale horse-urine, producing a purulent meningitis and meningo-encephalitis, and the symptoms of dumb rabies. Miller has reported three cases of seeming rabies in the cow, in which clusters of the parasite known as *strongylus tetracanthus* were found in the intestines. It is not unreasonable to suppose that the bite of an excited animal may be very dangerous, as it is an elementary fact in physiology that the secretions of the human female breast are at times fatal to the child. My friend, Dr. E. F. Brush, informs me that one of the earlier Borgias obtained a subtle and most dangerous poison by dipping a ring in the saliva of a hog which had been hung up and kept excited to a blind fury for a long time.

The variations are almost incredible that are alleged to exist in the period of incubation of hydrophobia. Fleming states that the average in England has been from thirty to fifty-nine days. According to Tardieu, it may vary from one month to twelve. Trousseau says that it may run from a few days to a year. In Algiers it varies from twenty to two hundred days. Sauvage speaks of a peasant who was suffering from the most pronounced hydrophobic symptoms on the third day after being bitten by a mad wolf. Chirac tells of a merchant who was bitten by a mad dog at Montpellier, afterward resided ten years in Holland, and was suddenly attacked by hydrophobia on his return to France, on learning of the death of a brother cadet who had been bitten by the same animal. Finco, of Padua, narrates the case of a young woman who became hydrophobic fourteen years after the bite of a rabid dog. Even Gowers states that the period of incubation is longer and more variable than that of any other known acute specific disease, and believes that many well-authenticated cases have occurred eighteen months after the wound. This extreme variableness in the period of incubation lends credence to the belief firmly held by many competent observers that death from fear can occur with symptoms closely resembling the true disease. These are the cases of so-called lyssaphobia or hydrophobia. Although a recent writer has characterized such a belief as childish, it is yet a matter of clinical observation that great psychical shock can produce mental disease and death. I have a patient in whom an attack of melancholia of eighteen months' duration was caused by her lover being suddenly thrown from his horse and killed before her eyes. I know of another patient to whom her husband, who had been supposed lost at sea, suddenly appeared, when she became speechless, mumbling, and, preserving only consciousness enough to caress his face gently for a few hours, sank into a coma and died. Another patient of mine with transverse myelitis, who had improved greatly, was much frightened by his room catching fire, and in a few hours he became delirious, and within twenty-four he was dead. I have had under my care a stalwart, resolute young man, to whom the Secretary of the Treasury has just presented one of those rare medals of gold that are given only to those who save a life at imminent danger to their own, and who, notwithstanding he had nerve enough to plunge into the raging surf to rescue a fellow-being, has not been able to do a day's work since, suffering from those well-known symptoms which Oppenheim describes as traumatic neurosis, and which occur so frequently after railroad accidents. (See "Railway Injuries," etc.) If these instances be not sufficient, a perusal of Tuke's book on *The Influence of the Mind on the Body* ought certainly to convince any impartial person that such an overwhelming fear as is that of hydrophobia is quite sufficient to cause death, the more especially as a number of suicides have been committed from this very dread.

Very few men in this city of New York, or, indeed, in this country, have seen a case of hydrophobia. Indeed, one of the great difficulties that I encountered in arranging a recent debate upon this subject in

the New York Academy of Medicine was that there was not a neurologist in New York City who had seen more than one case in his own practice, although some few had observed two or three cases at the great hospitals, while many men of great experience had never come in contact with any. For my part, in an experience of over twenty years as student and practitioner, I have never seen but one one case, and, to be guilty of an Hibernianism, I would not swear to that In the city of New York, from 1855 up to the present time, the number of deaths from hydrophobia have been as follows, according to the figures very kindly furnished me by Dr. John T. Nagle, viz., 1855, 4; 1856, 3; 1857, 2; 1858, 0; 1859, 2; 1860, 0; 1861, 5; 1862, 1; 1868, 3; 1864, 1; 1865, 3; 1866, 2; 1867, 4, 1868, 1; 1869, 5; 1870, 3; 1871, 7; 1872, 6; 1873, 0; 1874, 5; 1875, 0; 1876, 5; 1877, 4; 1878, 2; 1879, 0; 1880, 0; 1881, 3; 1882, 1; 1883, 1; 1884, 1; 1885, 0; 1886, 0; 1887, 1; 1888, 0; 1889, 1. Thus, in a period of time covered by thirty-five years there have been seventy-six deaths, in nine of these years there have been none, and it has twice happened that for two years at a time there was not one. One of the deaths in 1874 is that of a man who was bitten by a dog supposed to be rabid, and a coroner's inquest was held, the cause of death being stated to have been lyssaphobia. I come in contact every year with many physicians from different parts of the country, and it is very exceptional to have one tell me that he has ever seen a case of hydrophobia.

From this necessarily cursory review of the *pro* and *con* of the subject we may draw the conclusion, I think, that frequent mistakes are made in the diagnosis of rabies and hydrophobia; that death can be caused by the fear of the disease; that the symptoms of so-called dumb rabies may be caused by simple purulent meningitis and meningo-encephalitis; and that very few cases of either rabies or hydrophobia have been observed in New York City or in the country at large. Admitting all this, however, the question still remains as to whether there is a true rabies or a true hydrophobia. It all depends upon whether the virus obtained by Pasteur is capable of causing, when inoculated into a given animal, the same disease as was exhibited in the animal from which the virus was taken. Pasteur maintains that the poison of rabies is to be found chiefly in the medulla, spinal cord, and brain; that animals are made rabid generally by subcutaneous inoculation of this virulent nervous tissue, but that they are always made rabid by subdural inoculations after trephining; that rabbits when thus inoculated will develop a paralytic form of rabies, which has a period of incubation lasting from fifteen to twenty-one days, and that this paralytic rabies in rabbits is true rabies, as dogs inoculated with its virus develop true rabies. Pasteur claims that nearly all animals can be thus inoculated, although in the different species there are differences in the virulence and periods of incubation, the former being shortest and the latter longest in monkeys, and the former longest and the latter shortest in rabbits.

If the virus, however, is passed successively through a series of rabbits, first inoculating one and then waiting for the rabic symptoms

to appear, then inoculating the second from the spinal cord or medulla of the rabic animal, in each case making the inoculations beneath the dura mater, the period of incubation can be lessened finally from fifteen to eight days after passage through a series of twenty-five rabbits, and by further transmission through a second series of twenty-five rabbits it can be still further reduced to seven days, which seems to have been the limit of reduction. The virus is prepared by sterilizing in a sterilized bouillon or beef-tea portions of the medulla or spinal cord from rabid animals. The medulla or cord is removed with all possible care to prevent contamination from foreign matters, and is suspended in a jar, the air of which is artificially dried and subjected to a constant temperature of 20° centigrade. At the end of fifteen days this medulla or spinal cord will have entirely lost its virulence, and any degree of virulence may be obtained, therefore, by using a spinal cord upon any given one of these fifteen days. If the infectious cords, while in a moist condition, are submerged in carbonic acid gas and kept aloof from the air and its micro-organisms, they will remain virulent for several months.

In order to protect a dog from rabies, Pasteur inoculates in the following way: On the first day an inoculation is made with virus obtained from the cord of a rabbit in whom the incubation has been seven days, this cord having been rendered non-virulent by preservation as above for about fourteen or fifteen days. On the following day a cord of twelve or thirteen days old is employed; on the next day, one eleven days old; then on the fourth day, one nine days old; on the fifth day, one seven days old; on the sixth day, one six days old; on the seventh day, one five days old; on the eighth day, one four days old; on the ninth day, one three days old; on the tenth day, one two days old; and on the eleventh day, one one day old. By this means, it is claimed, the dog is finally rendered non-susceptible, so that no symptoms follow the last inoculation. These tests have been repeated and verified by Dowdeswell and Horsley in England, Shakespeare, Ernst, and Biggs in America, Hogyes of Buda-Pesth, Di Vertia and Zagari in Italy. Spitzka claims, however, as has already been stated, that he has been able to produce paralytic rabies in dogs by subdural inoculations of the spinal cord of a healthy calf, common yellow soap, and stale horse-urine, thereby producing a purulent meningitis and meningo-encephalitis. It does not seem to me, however, that these latter experiments should militate against Pasteur's, inasmuch as in none of the Pasteur cases was meningitis or meningo-encephalitis produced. Indeed, Pasteur and his associates do not claim any definite characteristic lesions for rabies. They simply maintain that in the medulla and the spinal cord there are found accumulations of round cells in the perivascular spaces, forming a so-called miliary abscess, and even these may not be found at all in the early stage, although in paralytic rabies in man they are more marked. Golgi states that the nuclei of the medullary nerve-cells are altered. Gibier describes a microbe in the brain of a rat, but he has not been able to color or cultivate it. Fol has demonstrated, in spinal cords of rabic animals, groups of small granules

resembling micrococci lodged in the neuroglia, or between the axis-cylinders and the sheath of Schwann. Cornil and Babes, however, have not been able to find these microbes of Fol. But Babes has found in the spinal cord of rabic animals round microbes in groups, which he has been able to cultivate, and the injection of which into animals produced rabies. He has also found another microbe in the cerebellum and spinal cord. This is very brilliant, and ordinarily forms dense and thick colonies resembling diplococci or ovoid bodies, often with a transverse striation in the middle. Rivalta and Dowdeswell have found certain microbes, but their conclusions have not been accepted by bacteriologists.

If we may trust the present writers upon the subject, the symptoms of furious rabies in the dog are as follows: Alteration in the dog's demeanor, so that it becomes sullen, shy, and restless; hallucinations of sight, so that imaginary objects will be bitten or snapped at; strange and erratic movements, indicative of mental aberration; transition from obedience and affection to semi-delirious outbursts, or to frenzy or fury; an irresistible tendency to roam, so that great distances may be covered in a surprisingly short time; great tendency to bite; greatly increased salivary secretion, which becomes thicker and more viscid; a peculiar howl, which is described by Fleming as being neither a bark, howl, growl, nor snarl, but a curious jumble of all, the howl being predominant; finally, exhaustion, paralysis, and death. In dumb rabies there is the characteristic paralysis of the lower jaw, so that the dog is incapable of either eating or drinking, or of closing the mouth; sometimes also a certain amount of tumefaction of the pharynx and tongue, and finally, paralysis of the extremities and death. In so-called tranquil rabies the animal is in a condition of coma. None of these animals show any aversion to water, contrary to the popular belief.

The symptoms in the human being consist at first of general *malaise*, mental depression, slight insomnia, and some vague distress about the throat. Attempts at drinking occasion pharyngeal spasm, which spreads to the muscles of respiration, causing a short, quick inspiration, like that produced by a sudden affusion of cold water, this increasing in severity to a strong inspiratory effort, in which the extraordinary muscles of respiration take part, so that the shoulders are raised, and even the facial muscles may draw the angles of the mouth outward. Reflex excitability is enormously increased, the slightest cutaneous impression bringing on a spasm. Salivary secretion is much increased. Vomiting is common. The muscular spasm, confined at first to the muscles of deglutition and respiration, may spread to the other muscles of the body, and the convulsions may become general, causing continuous muscular rigidity, or opisthotonus, or the convulsive movements may be co-ordinated and closely resemble the hysteroid variety. Delusions may occur, or even delirium. There is no such dread of water as to warrant the term hydrophobia, as many of even the recent writers would have us suppose; but the spasm of the muscles of deglutition at the sight of water is only a part of the general reflex excitability, as is shown by the fact that

glittering objects, loud noises, draughts of air, may cause the same spasms, or kindred ones, in other muscles. In many cases there is a curious alternation of consciousness with furious action. Thus, a patient begs the bystanders to go away, as he fears he may do them damage; or, as Dr. Ernst tells me that a patient of his did, a man may fly at the throat of an attendant, saying, "I can't help it!" The temperature ranges from 100° to 104° F., or, exceptionally, 106° F. Remissions are frequent. Death is sudden, or by gradual coma. Paralysis does not generally occur, although it has been stated that paralytic symptoms similar to those of acute ascending or Landry's paralysis may supervene; but as there are only two cases to support this claim, and as one—a man named Goffi—had been inoculated by Pasteur's "intensive" method, and died notwithstanding, it needs no eminent logician to perceive that the probability of the symptoms having been caused by Pasteur's intensive inoculation is much greater than the probability of their having been caused by rabies.

The differential diagnosis in the human being is from the following diseases:

Tetanus;
Delirium grave;
Acute mania;
Epilepsy;
Hysteria.

Tetanus has none of the cerebral symptoms.

Delirium grave is a disease of long duration, although in some cases it may be very difficult to make the differential diagnosis.

Acute mania has a more gradual onset on the one hand, or a more acute one upon the other, and has not the remissions or the reflex excitability of hydrophobia.

There should never be any question about the diagnosis from epilepsy—in which there is none of the reflex excitability, none of the remission, and in which there is, on the other hand, definite tonic or clonic convulsions, and the history of many cases of epilepsy (*vide* Chapter IX., "Epilepsy").

In hysteria there will be the history of the hysterical diathesis, or the peculiar emotional condition, or the hemianæsthesia; and sudden recovery from stout assertions of the absence of danger will confirm the diagnosis.

DIPHTHERITIC PARALYSIS.

History. Diphtheritic paralysis was first described by Lepois in 1580, again by Heredia in 1690, by Ghisi in the Italian epidemic of 1747 and 1748, by Chomel in France in 1748, and by Bard, of New York, in America in 1771. Singularly enough, these observations attracted but little attention until the early part of the nineteenth century, and since then it has been observed by all the prominent writers.

Clinical Symptoms. The paralysis is usually not observed until the first or second week after the convalescence from diphtheria,

although it may occur earlier or later. It is generally preceded by some of the usual buccal symptoms, although they may be very slight, but it has been claimed that it may occur without them, and Boissarie has related several such seeming cases, which are certainly rare enough to be worthy of note. The most common symptoms are loss of the knee-jerk and palatal paralysis. The latter is evidenced by the nasal intonation, slow speech, snoring in sleep, impaired deglutition, and nasal regurgitation of liquids. In such cases there are usually a sensory paralysis of the fauces and uvula and loss of the reflex. Multiple paralysis is the next most frequent form, and this may be preceded or not by the palatal paralysis, although it is usually preceded by the loss of the knee-jerk. Different muscles may be affected. The limbs are most frequently implicated, the paralysis is usually bilateral and symmetrical, and occasionally there is some impairment of sensation; the trunk-muscles may also be affected. Cardiac disturbances are comparatively infrequent, but they are of very grave import when they do occur. Hemiplegia is very rare. The bladder is affected in only very severe cases. The most common symptom due to implication of the eye is impairment of near vision, due to paresis of accommodation, and with this is sometimes associated a paralysis of the internal muscles of the eyeball or of the internal recti, and even ptosis. Almost all the senses may be affected in different cases, although impairment of hearing never occurs. Convulsions occur occasionally in severe cases. After the paralysis has lasted for several weeks there is generally a decreased faradic reaction, and this may proceed to entire loss of response to this current, although the muscles ordinarily respond to the galvanic current. There may even be a reaction of degeneration.

Pathological Anatomy. To the naked eye there are seldom any morbid appearances except some hyperæmia or minute extravasations. Microscopically the muscles are sometimes normal, and in other cases they present granular and fatty degeneration. In the nerves degeneration is found in all the severe cases, either in the peripheral portions or in the whole trunk, although the posterior roots are usually normal. The medullary sheath breaks up, the nuclei of the sheath of Schwann multiply, and occasionally the axis-cylinders disappear. The interstitial tissue is generally unaffected. These changes have been found both in the palatal nerves and in those of the limbs. The changes are usually limited to the peripheral nerves, but occasionally the cells of the anterior cornua of the spinal cord undergo granular and fatty degeneration, or are swollen, atrophied, with degenerated processes, or free nuclei are found in the gray substance. Occasionally, though rarely, the walls of the vessels are found thickened, or the vessels are dilated and congested. There is sometimes softening of the substance of the cord, and in other places a hyperæmia without softening, in conjunction with the cellular changes that have already been noted. The pathological appearances differ very much in different cases, so that they may vary from scarcely any change to the most marked of those which we have

detailed. The truth of the matter seems to be that the disease is dependent upon the capricious action of a micro-organism, so that sometimes one lesion is found, sometimes another, sometimes one portion of the nervous system is affected and sometimes another, and it has even been observed in certain cases that a single branch of a nerve may lose its function while the other branches are unaffected.

The micro-organism, however, is still a matter of doubt. At one time it was supposed that it was the micrococcus or spherical bacterium; others have stated that the diphtheritic micrococci were the same as the septic micrococci. Curtis and Satterthwaite, reporting to the New York Board of Health in 1877, affirmed their belief that the micro-organism could not be distinguished from that which occurred in the ordinary septic material, as well as that it was the chemical substance or ptomaïnes produced by the diphtheritic micro-organisms that caused the usual symptoms. Wood and Formad, in 1882, confirmed these statements. In 1883 Klebs found a peculiar bacillus in the pseudo-membrane and in the surrounding inflamed tissue, and this was afterward investigated by Loeffler, and has since been known as the Klebs-Loeffler bacillus. This bacillus is motionless, straight or curved, about the size of the tubercle bacillus, but doubly thick. Whilst it is found in the membrane, it is not found in the lymphatics, internal organs, or bloodvessels. Loeffler, however, was not able to find this bacillus in some typical cases of diphtheria, although Wellenhof has it. Oertel states that there are two kinds of microbes, namely, chain-forming cocci (streptococci) and rod-shaped bacteria with rounded extremities (bacilli), the former being found in the pseudo-membrane, the latter in the fibrinous networks. Prudden goes no further than to say that there is a streptococcus almost constantly present in the pseudo-membrane. M. W. Taylor, of London, has seemingly adduced proof to show that common mould may be a cause of diphtheria, as several cases occurred in a sleeping-room whose walls were wet, and from which were taken the aspergillus, coprinus, and penicillium moulds.

Prognosis. The prognosis of diphtheritic paralysis is usually good, although cardiac symptoms, weakness of respiration, or long-continued difficulty of deglutition may make it a serious matter.

Diagnosis. The diagnosis of diphtheritic paralysis must be made upon the history of a paralysis having succeeded a throat trouble; or if this is not possible, by the ocular symptoms conjoined with the absence of knee-jerk and inco-ordination that have come on within a brief time. Hysteria is said sometimes to simulate diphtheritic paralysis, and I can imagine that this might occur, but I have never seen a case. The diagnosis can easily be made by the fact that in hysteria there is not the atrophy, the ocular symptoms, the loss of knee-jerk, or the partial paralysis and loss of reflex.

Treatment. The treatment of diphtheritic paralysis should be by rest, careful feeding, drugs, and electricity. The patient should be put to bed if the paralysis involves any important nerves or muscles, or if it is in the neighborhood of such. Even if the muscles affected are not of great importance to life, it is certainly a good rule to keep

the patient in bed until it is determined exactly how far the disease is going to progress, for the first symptoms are not necessarily the severest ones. The diet should be of the most nutritious character, and suitable to the age of the patient. It should always be fluid where there is any difficulty in deglutition, and with little children the best food is milk, with farinaceous substances and nutritive jellies. With children of more advanced age milk should always be the basis of the diet, and to this should be added either a home-made beef-tea, or some one of the beef-preparations, and the meat should be cut up into minute particles or the juice squeezed from meat. With adults, milk, farinaceous substances, nutritive jellies, beef-tea or beef-extracts, and koumiss, will form an abundant diet so long as there is danger of failure of deglutition. As soon, however, as the difficulty of deglutition recedes, the diet should be rapidly pushed to the highest point of nutrition that is possible, and there is no danger whatever of too much food, provided that the skin and the bowels act fairly well. It is also generally advisable to employ alcoholic stimulation in adults, and the amount used should vary according to the age and habits of the patient; but, as a rule, the quantities will range from 1 to 3 ounces in the twenty-four hours. When there are cardiac symptoms the patient should be kept in a recumbent posture and digitalis should be administered, the dose varying in a child from 1 to 5 drops of a good, freshly and reliably prepared tincture every four or five hours, or $\frac{1}{2}$ to 2 drops of a trustworthy fluid extract at the same intervals. Alcohol is also of considerable value in doses of 5 to 10 drops in a child to a teaspoonful for an adult, every two or three hours. Use should always be made of the aromatic spirit of ammonia when dangerous cardiac symptoms suddenly supervene, and in diphtheritic paralysis of gravity it is my custom to have this aromatic spirit of ammonia kept at hand in case of an emergency arising. The sulphate of strychnine is also a very useful drug in cases of cardiac paralysis. These four substances—digitalis, strychnine, alcohol, and spirit of ammonia—should be used with a method, however, and not indiscriminately. The ammonia has only a temporary effect, and should therefore be employed until the other drugs can begin to act. The alcohol will act most quickly next to the ammonia. The strychnine and digitalis begin to act at about the same period of time. For these reasons the ammonia need only be used at the onset of the symptoms, or in case of a relapse, whilst the others should be continuously used until danger is past. The sulphate of strychnine is best administered hypodermically, and the physician, during a case of diphtheritic paralysis, should have a solution at hand; $\frac{1}{100}$ grain in a very young child, $\frac{1}{60}$ grain in one older, or in an adult $\frac{1}{30}$ grain usually will be sufficient, injected once a day. When the immediate danger of any fatal result has passed, attention may be directed to arresting the degeneration of nerve and muscle, and the best means of doing this are massage and electricity. Massage must be used very gently, only upon the affected muscles, and never prolonged over ten or fifteen minutes, and it should be borne in mind in giving it that the object is to facilitate the passage

of blood through the muscle with the least possible mechanical irritation. Faradism should be applied gently to the muscles and nerves affected, the current at first being used only sufficiently to be just felt by the patient, and then gradually increased, but never made painful, and the sittings at first should not be over three to five minutes, and increased by degrees to ten or fifteen minutes. At this stage, also, tonics should be administered, and the best of these, in my opinion, is a tonic of bark or quinine, strychnine, and iron. The ordinary citrate of iron and quinine of the Pharmacopœia is as good as any, and the dose should be 1 grain three times a day to children, 3 grains three times a day in adults; and with this may be combined sulphate of strychnine, $\frac{1}{100}$ grain three times a day in infants, $\frac{1}{60}$ in children, and $\frac{1}{30}$ in adults, also three times a day. If children cannot take pills, or if there is difficulty in giving them any medicine that has much taste, the quinine may be given in the form of the tannate in chocolates, 1 or 2 grains each. The iron may be given in the form of the subcarbonate of iron, 10 to 30 grains, according to the age, and this may be put into some hot milk darkened with tea, or into a cup of cocoa, where its taste will hardly be perceived, and the strychnine may be given in solution. All these substances, however, may be administered almost as well in the form of the tablet triturates which are made up nowadays so reliably by many manufacturing houses. As the patient progresses into convalescence, treatment should not be abandoned, but tonics, in some cases small quantities of alcoholic stimulants, abundant and varied diet, and electricity, should be continued, and every effort should be made to avoid fatigue. These patients often remain weak for a long time afterward, and they must be subjected as little as possible to fatigue and depressing influences. Even after a recovery has been made there will often be left awkward postures from muscular paresis, and sedulous efforts should be made to overcome these by the use of massage and gymnastic exercises, the latter being directed to exercise and strengthen the affected muscle or muscles. Braces or mechanical supports, however, should not be used, because their tendency is to cause the patient to rely upon the steel or iron of them rather than to increase the strength of the paretic muscles.

CHAPTER XI.

CERTAIN NERVOUS SYMPTOMS COMMON TO DIFFERENT DISEASES.

VERTIGO.

Vertigo is a symptom of so frequent occurrence and its import is apt to be so much exaggerated that I have thought it wise to devote a special section to the subject. The meaning of the word should be precisely understood. By vertigo is meant a subjective sensation of loss of equilibrium without loss of consciousness. It has been the custom of late years to employ the term in certain losses of consciousness, notably in the so-called laryngeal vertigo, which is really a laryngeal epilepsy. In true vertigo consciousness is very seldom lost, although there may be a momentary obscuration of consciousness. The causes of vertigo are manifold, and they may be catalogued as follows:

Lithæmia;
Aural disease;
Eye-strain;
Anæmia;
Alcoholism;
Gout;
Nephritis;
Intracranial lesions;
Spinal lesions.

Of all these causes the most frequent in my experience is lithæmia. Gowers states that aural lesion is the most common cause, but his experience has either been contrary to mine, or else the types seen in England are different from those observed in this country. The vertigo of lithæmia, as has been described in the section upon "Neurasthenia," may vary from a slight sensation of loss of consciousness to a dizziness that causes a person to reel almost as if intoxicated. The diagnosis of this form of vertigo can easily be made by attention to the symptoms that have been narrated when speaking of lithæmic neurasthenia, as well as by an exclusion of other causes.

Aural lesion I should classify as the most frequent cause of vertigo next to lithæmia. The lesion may be either in the external auditory meatus, the middle ear, or the internal ear, and the diagnosis between these different localities will be found stated in detail in Chapter XV., "Ménière's Disease." As has been seen, the auditory nerve terminates peripherally in the internal ear, thence runs up to the pons, to the nuclei in the floor of the fourth ventricle, and thence to the cerebellum, whilst its cortical termination is in the

posterior portions of the first two temporal convolutions. It is still uncertain as to whether lesions along the trunk of the nerve are capable of causing vertigo. Dana has related a remarkable case of lesion of the temporal lobe with great disturbance of equilibrium, but he was himself uncertain as to whether there was not trouble in the middle or internal ear. It has not yet been proved that the disturbance of equilibrium of cerebellar disease is due to the implication of the auditory nerve, and there is very much to contradict any such view. It is generally no difficult matter to make the diagnosis between a lesion of the ear in its external, middle, or internal departments, and a lesion of the nerve-trunk or of the base of the brain or the cerebellum or the temporal convolutions. (See chapter on "Localization of Lesions," etc.)

Nephritis is sometimes a cause of vertigo, but the diagnosis can be readily made by a qualitative, quantitative, and microscopical examination of the urine, although it must be remembered that a small quantity of albumin or a few casts may be found with various dietetic and functional anomalies.

Eye-strain has been generally supposed in the profession to be a very common cause of vertigo, but this has not been my experience. It is undoubtedly sometimes a cause, but not nearly so often as is supposed. This eye-strain may be due to strabismus, errors of accommodation, or exophoria.

Anæmia is sometimes, though rarely, a cause of vertigo, and in all doubtful cases not only should the heart and the great bloodvessels coming off from the heart be carefully examined and auscultated, but it may even be necessary to examine the blood.

Alcoholism, like anæmia, is sometimes, though rarely, a cause of vertigo, and the diagnosis cau be made with ease.

What has been said of anæmia and alcoholism is also true of gout.

Intracranial lesions of various kinds may be the cause of vertigo, but this is seldom the case unless the intracranial lesion is pronounced. This fact should be impressed upon patients, because it is popularly supposed that vertigo means intracranial lesion, and I have again and again seen lives blighted from a fear of this and the doubt of physicians. It is easy enough to make the diagnosis by the presence of the classical symptoms of intracranial lesions, namely, vomiting, pronounced headache, optic neuritis, optic-nerve atrophy, paralysis, motor or sensory, such defects of vision as hemianopsia or amaurosis, word-deafness, aphasia, and mental impairment.

Lesions of the spinal cord are infrequently the cause of vertigo, except in the case of locomotor ataxia. The diagnosis must be made by the presence of the well-known symptoms of spinal lesions, such as paralysis of motion and sensation, the bladder and rectum, atrophy, loss of tendon-reflex, violent pains, etc.

In the vast majority of cases, however, vertigo is due to lithæmia, aural disease, or eye-strain; indeed, so much is this the case that it is usually safe to assume that one of these three causes is at play

when a patient comes complaining of vertigo, for when there are graver lesions the vertigo becomes a minor symptom in the patient's estimation.

The prognosis of the vertigo will depend upon the lesion. In lithæmia it is very apt to be persistent, especially during the warmer months of the year, and it may continue for years or throughout a lifetime, so that it is well to inform the patient of this fact, and induce him to become accustomed to the symptom and to regard it as of no importance. In the section upon "Neurasthenia" I have spoken more fully upon this subject. When the vertigo is caused by aural lesion the prognosis will depend upon whether the cause is removable, which is generally the case in lesions of the external ear, often the case in lesions of the middle ear, and seldom the case in lesions of the internal ear. When the vertigo is due to eye-strain the prognosis varies. If there is exophoria, or strabismus, or error of accommodation that can be removed, the prognosis is good; but when it is due to amaurosis or hemianopsia, the causes are usually not removable, and the prognosis is grave. The vertigo of anæmia is of excellent prognosis. When due to alcoholism it is usually curable if the alcoholism can be cured and has left no structural lesions. The vertigo of nephritis depends upon whether the nephritis is curable, so that the prognosis is good in the acute nephritis of children after the exanthemata, and also often good in the acute nephritis of adults, whilst it is grave in the chronic forms of nephritis. In intracranial and spinal lesions the prognosis of vertigo depends upon whether the spinal lesion is one that can be cured. Usually, with the exception of intracranial syphilis, intracranial lesions are of grave prognosis, so that the vertigo resulting from them is a serious matter, and this is also true of spinal lesions capable of causing vertigo.

For the vertigo itself there is no treatment except the removal of the cause.

HEADACHE.

Like vertigo, headache is so general a symptom that I have thought it best to devote a short section to it. The causes of headache are—

- Migraine;
- Eye-strain;
- Nephritis;
- Anæmia;
- Alcoholism;
- Lithæmia;
- Reflex headache;
- Melancholia;
- Acclimation;
- Indigestion;
- Intracranial lesion;
- Locomotor ataxia.

Migraine, when typical, is a quasi-periodical headache limited to

one side of the head or spreading over the whole head; but when it is non-typical it may be more or less localized. The typical migraine can be easily diagnosed, because it comes only at intervals, lasting a day or a few days, between which the patient is entirely free from the attacks. The non-typical or localized migraine, however, is often not properly recognized, and more especially is this apt to be the case when the typical attacks of migraine have become less in degree or have largely died out, and their place has been taken by a constant localized headache or neuralgia. Migraine, it should be remembered, is a very mobile disease, and it is by no means infrequent, as I have pointed out in the chapter upon it, to find its place taken by epilepsy, and it may also disappear and lose its typical periodicity and intensity. The diagnosis, however, can be made by any one who is aware of this circumstance, for all that is necessary is to inquire carefully into the antecedent history, when the patient will be found to have had previous attacks of migraine; and to some extent the diagnosis will be confirmed by the success of the proper treatment.

Eye-strain is sometimes a cause of headache, but not nearly so often as physicians generally suppose. Errors of refraction, strabismus, and exophoria may cause it. The diagnosis can be made by exclusion of other causes and by the determination of the causes capable of causing eye-strain.

Nephritis is a frequent cause of headache, and in every case of headache occurring in an adult that cannot be properly explained a careful qualitative and quantitative chemical analysis should be made of the urine.

Anæmia is also a frequent cause of headache, and the diagnosis must not be made by observation of the color of the lips and the conjunctiva, but the heart and large vessels leading off from the heart should be carefully anscultated, and, if necessary, a microscopical examination should be made of the blood. The diagnosis is readily made.

Alcoholism is not a very frequent cause of headache, but it does sometimes produce it.

The headache of lithæmia is not mentioned here because it is a true headache, but rather because I desire to call attention to the fact that it is more often a sense of pressure and fulness about the head.

Headache may sometimes occur reflexly from disease of non-nervous organs, such as the stomach and the pelvic viscera, the latter especially in the female; but this cause is by no means so frequent as is generally supposd.

The headache of melancholia, like the headache of lithæmia, is not mentioned here because it is a true headache, but rather to call attention to its distinction from a trne headache. The melancholiac suffers only from various abnormal sensations about the head when he is afflicted with what I have called simple melancholia, and in snch a case the sensations are mainly or entirely in the back of the head and neck, but they will sometimes pass up to the vertex and

diffuse themselves over the head, and they may even occasionally be intense. The accompanying melancholia, the peculiar *facies*, the insomnia, and the suicidal impulse will, as has already been stated (*vide* "Melancholia"), make the diagnosis easy.

The process of acclimation will often produce vague, diffused headache that may at times become quite severe, and this process of acclimation, as is often overlooked, is not only observed in those who take up a permanent residence in a place, but in those who go to a different climate for a short time. It is constantly seen in the mountains and at the seashore during the summer movement of our American population, and in a less degree in the winter resorts. Nor is it observed only in those who have made a great change of climate, for even the dwellers in our seaboard cities suffer from it in first taking up their residence at the seashore. It is generally, but erroneously, ascribed to errors of digestion, although often no proof whatever can be obtained of the existence of these.

Indigestion is sometimes a cause of headache, but not nearly so often as is generally supposed.

Intracranial lesions causing headache can, of course, be readily diagnosed by means of the characteristic symptoms, such as neuro-retinitis or optic-nerve atrophy, paralysis of motion and sensation, word-deafness, aphasia, blindness or partial defects of vision, vomiting and convulsions. The headache of intracranial syphilis is, as I have already stated ("Syphilis of the Nervous System"), a quasi-periodical headache occurring either toward night or during the afternoon, sometimes in the morning, and is accompanied by obstinate insomnia, and the headache and the insomnia cease upon the supervention of paralysis or convulsions, or dementia—*i. e.*, upon the supervention of destructive lesions.

Of all the spinal lesions, locomotor ataxia is the only one that will give rise to headache, and this is only exceptionally. When it does occur it is an intense burning, stabbing, or lightning-like pain that either comes and goes with great violence and caprice, or in some exceptional cases is localized. The diagnosis, however, is readily made by the coexistence of other symptoms of locomotor ataxia, such as the characteristic ataxia, stabbing and lightning-like pains in the limbs and trunk, the loss of knee-jerk, the optic-nerve atrophy, and the bladder-symptoms.

The prognosis of headache will depend, of course, upon the cause. That of lithæmia, eye-strain, alcoholism, anæmia, acclimation, and reflex disorders is good. The headache of nephritis is of serious omen, unless the nephritis is an acute and transient one, such as occurs in children after the exanthemata, or in some cases in adults. The headache of intracranial lesion is usually a serious matter, except in the case of intracranial syphilis in the early stages, before structural lesion has taken place, as indicated by paralysis and convulsions, when it may be entirely cured by vigorous and proper treatment. The headache of locomotor ataxia is of grave prognosis, although it may often be alleviated considerably.

The treatment of headache depends upon the cause to a certain

extent, for if this is removable, such removal may be all the treatment that is required. For the relief of the pain itself the best drugs are opium or morphine, antipyrin, phenacetine, the bromides, cannabis indica, menthol, and cerebral galvanization. Opium, or, better still, its alkaloid, morphine, is undoubtedly the best drug with which to relieve violent and acute headache, and the physician should not hesitate to employ it in proper cases. He should always administer it himself hypodermically, however, and never trust the hypodermic needle in the patient's hands, nor should he give it by the mouth, not only because it is far less efficacious than when given hypodermically, but also because there is great danger of the patient forming the morphine-habit. It should be laid down as an axiom that a headache must be controlled and that the headache-habit must be broken up, and, if necessary, this must be done by means of morphine. In most cases, however, it will not be necessary to resort to this drug, which, although it is undoubtedly the most efficacious, may yet lead to the formation of a dangerous habit. It is a curious clinical fact that although morphine or opium may be given with impunity in melancholia, so far as the formation of the habit is concerned, it is very apt to lead to the formation of such a habit when used in simple headache unless there is a grave necessity. In other words, it would seem as if great pain or grave disease protected the human being against the formation of the morphine-habit. Antipyrin undoubtedly stands next to opium in its effect upon the headache, but the objection to the drug is that it is apt to be depressing, and it should therefore be employed with great caution. It is best to give it in doses of 5 grains every hour or two until the headache has been controlled or until 15 or 20 grains have been given, watching the pulse carefully all the time, and ascertaining by the experience of one attack what the patient can stand in the next. Phenacetine is not a reliable drug in cases of headache, as it has in my experience a more peculiarly beneficial effect upon the nerves of the trunk and limbs than upon the nerves of the head and neck. Nevertheless, in some exceptional cases it will act very well, and it is always worth while to make a trial of it if the headache is not very acute, as it produces very little depression, so that doses of 15 to 30 grains may be given to a healthy individual of average strength in the course of the twenty-four hours. The bromides are not of much use except in the headaches due to acclimation and migraine, in which they sometimes act like a charm, and occasionally they will do very well in the headache of eye-strain, lithæmia, nephritis, and some headaches that are due to intracranial lesions. Cannabis indica is seldom of any use except in the headache of intracranial lesions and migraine, but in the latter it must be given in continued doses of $\frac{1}{3}$ or $\frac{1}{4}$ grain between the attacks, and during the attacks opium or morphine, bromide, antipyrin, or phenacetine must be employed. Peppermint in doses of 10 to 15 drops of spt. menth. pip. (U. S. and B. P.) every hour or two, in a wineglass of water, or in the form of a menthol-pencil rubbed over the aching head or brows, is sometimes of great use in the lighter forms of headache, but it is absolutely worthless in severe cases. Cerebral galvaniza-

tion is only of use in the cases of slight continuous headaches, and it is valueless in the acute attacks. A small current of two or three milliampères, with a sitting of three minutes, repeated every day or several times in the day, should be employed. Combinations of these different drugs may often be made with advantage. Thus, in cases of migraine a combination of cannabis indica with bromide of potash and phenacetine will often act like a charm, as in this prescription :

℞.—Potass. bromid.	ʒj.
Phenacetine	℥ss.
Cannab. indicæ	gr. jss.

M. ft. in chart. no. vj.
S.—Dose, one powder in a wineglass of water ; to be repeated as needed.

Or in the headache of intracranial lesion a combination of cannabis indica, bromide of potash, and sulphate of morphine, will also act beneficially, as thus :

℞.—Ext. cannab. ind.	gr. ij.
Potass. bromid.	℈iv.
Morph. sulph.	gr. j.

M. ft. in chart. no. viij.
S.—One powder once or twice daily, in a cup of milk, beef-tea, or cocoa.

Sulphate of quinine may be combined with some of these drugs with great advantage in certain headaches, such as those of intracranial lesion, locomotor ataxia, acclimation, nephritis, and intracranial syphilis. It is a curious clinical fact, however, that the headache of intracranial syphilis cannot be controlled by any of these analgesics, and the best remedy for it is the iodide of potash in the large doses that have been recommended in the section on "Syphilis of the Nervous System." When the headache is acute, quiet, rest, cold or warm applications, as may be most agreeable, will be useful adjuvants, and at such periods a brisk movement of the bowels or even an emetic will also aid greatly. In headache from indigestion a full dose of a reliable pepsin will often give remarkable relief when the indigestion is stomachic, but when it is intestinal 5 grains of salol will act much better than pancreatin. The salol may be repeated three or four times during the day if necessary. The headache of anæmia can be overcome not only by the administration of large dose of iron (*vide* "Neurasthenia"), but it should be remembered that this will often not be sufficient, and in such cases sulphate of quinine, 2 grains three times a day, with $\frac{1}{32}$ grain of the bichloride of mercury, should also be administered, and care should be taken that the bowels are kept in good order, although there should be no violent purgation or excessive use of mercurials. The general health in all cases of headaches should be carefully attended to. The diet should be as nourishing as possible, and great attention should be paid to its variety. Even in cases of headache due to indigestion the patient should not be allowed to enter upon the Sisyphus-like task of eliminating those articles of food which are thought to cause indigestion, for experience has shown me that

systematic elimination of this kind very often leads to a downright process of starvation. The details of such a feeding process have been fully dwelt upon in the section upon "Neurasthenia." Indeed the treatment of the different varieties of headache should be studied in the different chapters in which I have discussed the various causative lesions.

INSOMNIA.

At the very outset of this subject we are met by the query as to what we know of the exact molecular alterations in the brain that constitute sleep, and to this our answer must be absolutely negative. There have been all sorts of theories, to be sure, as to the *rationale* of sleep, but they have been pure theories. Durham, Hammond, Mosso, Veit, Pettenkoffer, and others have thought that sleep was due to anæmia, or, more properly speaking, to ischæmia of the cerebral substance, or to a diminished use of oxygen in the cortical cells. But no one has made the slightest effort to show whether these phenomena are cause or effect; nor is it possible in the existing state of our scientific apparatus to know anything more, for the molecular changes that take place in purely functional phenomena can never be known until we have reached such a point in our scientific development as to be able to plunge some instrument into the brain and study the action of the living cell. It must be remembered that the cortex of the brain, some alteration in which probably produces sleep, is a wonderfully complex structure. Seven or eight cortical layers have been described, each differing as much from each other as do the cells of different organs; and we know absolutely nothing of the function appertaining to each layer of cells, except that there is a probability that certain of the larger ones—the so-called ganglion-cells—are possessed of nervous functions.

At the very beginning, therefore, we must confront squarely the unwelcome fact that sleep and its derangements must be studied from a purely clinical and therefore somewhat empirical point of view, and that in order to combat insomnia intelligently we must study the conditions of disease in which it is manifested. Limiting ourselves in this manner, we find that insomnia is caused by—

The passions, especially worry, anxiety, and grief;
Pain;
Febrile disorders;
Neurasthenia;
Certain insanities;
Certain organic diseases of the cerebrum;
Certain drugs or food-substances.

The treatment of insomnia will vary very greatly according as it is being produced by one or the other of these causes, with which it will be best to deal separately.

At this point it may be well to dwell for a moment upon the means at our disposal for inducing sleep, and especially upon the different hypnotics now in vogue, namely: chloral, sulphonal, trional, chloralamid, urethan, and paraldehyde. I have ranked these in the

order in which I consider them the most efficacious. In my hands chloral hydrate is the most effective of them all; but it is a drug which I rarely employ, because of the depressing effects it is likely to produce, especially if long continued. Sulphonal is the drug which has acted best, by all odds, in my hands, although I seldom give it alone, but usually in combination with bromide, or, as I shall have occasion to indicate later on, with opium. Ten grains of sulphonal, with 10 or 20 grains of the bromide of potassium, are an effective sleep-producer in the majority of cases, and the dose of sulphonal can be increased, if necessary, to 20 or 30 grains, although it is seldom necessary to give more than 20 grains of bromide. Sulphonal should always be given about an hour before bedtime, best in a cup of bouillon, soup, milk, or chocolate, or in a compressed tablet. In my experience it is not absorbed for an hour or so, and its maximum effects are produced three or four hours after it is taken; so that it is not a reliable drug to produce sleep quickly, for which purpose chloralamid is preferable. The dose of chloralamid should be much larger than that of sulphonal, never less than 30 drops at the outset, and frequently 40 to 60. The objection to it is its taste, which can be disguised by giving it in capsules or tincture made with some bitter substance. Lately, however, it is made into compressed tablets, which are the best means of administration. In many cases, when it is necessary to induce sleep immediately, and when the sulphonal alone or with the bromide will not do this, I use chloralamid first, and then instruct the patient to take the sulphonal when drowsiness is beginning. Trional is fairly reliable in some cases, but it is capricious in its effects, and much more apt to produce unpleasant results. Urethan in my hands has not been a reliable drug except in the very mildest forms of insomnia. Paraldehyde is ranked by some authors as a very effective hypnotic, but I have not found it reliable, perhaps because my use of it has been limited on account of the very unpleasant, mawkish odor of the breath that it is apt to induce in certain patients when continued. This odor cannot be removed by any means that I have tried, although the tincture of bitter orange-peel has been vaunted against it—which, however, I have found to be perfectly useless. Aside from the use of these hypnotics, pure and simple, we can in some cases induce sleep by mental and physical repose, by hypnotism, by cold and warm effusions, by electricity, by change of scene and surroundings, by travel, by massage, and by stimulants.

Probably the most intractable of all forms of insomnia is that caused by the passions. In such cases it becomes a matter of infinite tact to know how far it is best to use a pure hypnotic and to what extent it is wise to accustom the patient to the use of such drugs. Usually, however, sleep can be at least temporarily induced by the employment of the bromides in combination with one of the pure hypnotics. Isolation that shall conduce to mental and physical repose will, of course, be of very great aid; but, after all, there is no means, save time, of lessening the perturbation of a great passion.

The insomnia produced by pain usually proceeds from neuralgia,

neuritis, the early stage of pleurisy, pneumonia, peritonitis, and certain acute pelvic affections in the female, articular and muscular rheumatism, certain painful affections of the eye, certain infectious disorders (especially influenza), intracranial tumor, cerebral meningitis, and intracranial syphilis. In all these conditions the rule should be to ascertain first how far the pain can be controlled by the proper treatment of the disease, and then how much sleep will come when the pain is relieved. In no case, however, should analgesics and hypnotics be used except as adjuvants to the proper treatment of the disease directly causing the pain and indirectly the insomnia. Then the next consideration should be as to whether the pain is acute, subacute, or chronic; for while it may be permissible, and often necessary, to use analgesics and hypnotics for the relief of pain that is acute and of short duration, it might be very inadvisable to use them in chronic cases. In one disease, however, intracranial syphilis, it is perfectly useless to attempt to overcome the pain or the insomnia except by means of iodide, with or without mercury, and this should never be forgotten. In neuralgia the cause of the pain producing insomnia should always be carefully sought for, and it is generally to be found in a condition of malnutrition or some direct or indirect irritant. In the case of malnutrition the pain and the resulting insomnia should first be treated by means of rest and abundance of good and varied food, with milk and beef-tea (*vide* section on "Neuralgia"). Every case of neuralgia that is not due to a direct or reflex irritant should be treated by means of such combination of rest and dietetics, to which in many cases it may be useful to add iron, either the dialysed or the albuminate, in 2 or 3 drachm doses, three times a day, given in a cup of water after meals, or the peptonate of iron, in compressed tablet, gr. iij three times daily, or ferratin, gr. iv–viij, three times daily, in capsule. But if neuralgia is due to a direct or reflex irritant, it will have been caused by either a neuroma, a cicatrix, a mass of callus, an inflamed joint, a pulmonary or abdominal adhesion, an inflamed viscus, a lesion of the naso-pharynx, of the ear, or of the eye (either error of refraction or muscular insufficiency or direct ocular lesion), nephritis, lithæmia, gout, anæmia, or leucocythæmia; and all of these should be carefully sought for and treated, if present, before the pain and insomnia are directly attended to. In many of these cases, therefore, it will happen that the pain and subsequent insomnia will cease without the direct need of any analgesics or hypnotics; but if this should not be the case, it may become necessary to relieve the pain absolutely, and this should be done by means of quinine in 2 or 3 grain doses, in combination with a reliable and fresh preparation of salicylate of sodium in 2 or 3 grain doses, two or three times a day; or, if this will not answer, phenacetine may be added, 5 or 10 grains at a dose; or it may even be advisable to try antipyrin, in doses of 10 to 15 grains, but I am very much opposed to this drug because of its depressing effects, of which I have seen some sad examples, so that I should not advise its use even in the most robust individuals, except temporarily. Blisters along the course of the affected nerve, or

occasional cauterization with the Paquelin cautery, will often be found of great value. Exalgine has not proved reliable in my hands. If morphine is to be used, it should only be for the control of acute pain that cannot otherwise be relieved, and then it should never be given except by the physician himself. I am not one of those who place much credence in the sensational stories about the formation of opium-eaters by physicians, for I believe that when a man or woman becomes an opium-fiend, in the vast majority of cases there is an original defect in that man or woman which would have led them to form this or some other habit upon favorable opportunity. I know of no reason why all of us cannot steal or forge or take opium or hasheesh or gamble or lie or do other things which the moral sense of civilization condemns, unless it is that we have within our brains and spinal cord that restraining force which physiologists call inhibition and theologists call conscience; and the individual who forms the opium or any other drug-habit is usually one of those afflicted with an hereditary or personal predisposition toward vicious habits. I should therefore not use opium in one of this class, but I should employ it freely for the relief of acute pain in one who is not of this type. If hypnotics are needed, after all, sulphonal or chloralamid is usually the best. Of neuritis the same remarks may be made as of neuralgia.

In the early stage of pneumonia and pleurisy the pain and insomnia may often be relieved by hot fomentations, an oil-skin jacket, and rest in bed, without resort to analgesics or hypnotics; and either analgesics or hypnotics should be administered with very great regard to the condition of the heart and extent of implication of the lungs; indeed, in all cases it will be best, if possible, to rely upon the effects of stimulation by means of eggnog, milk-punch, and some light food-substance at bedtime, rather than upon pain-relievers or sleep-producers. If hypnotics have to be used, sulphonal and chloralamid are again the best.

In peritonitis opium can be used with a far freer hand than in the pulmonary affections, for the well-known reason that it restricts the friction of the peritoneum, and it may be quite sufficient to relieve the insomnia. If not, hypnotics must be used, and I know of no particular choice among them except for the general reasons that have been stated in the introductory paragraph.

These same considerations apply to the treatment of acute pelvic affections of the female.

In acute articular rheumatism the salicylates, alkalies, lotions, and rest will usually be found to be of more value for the relief of pain than morphine or the other analgesics; and therefore, if the insomnia is not relieved by the former means, use should immediately be made of the hypnotics proper, but these, of course, should only be employed so long as the cases are acute, and should be withdrawn as the disease becomes chronic, when the continuance of the insomnia should be met with some light food or stimulant; or with massage or gentle faradization at bedtime. The insomnia caused by the pain of articular rheumatism should never be treated by morphine or opium unless

it is very acute, when it may have to be met temporarily in this way; but in the vast proportion of cases hot applications, galvanization, faradization, massage, salicylate of sodium, oil of gaultheria, and rest, will suffice to overcome the insomnia if the causative disease is properly treated.

In some acute affections of the eye the pain may be so sharp as to cause insomnia, and in such conditions the relief of pain by means of cocaine will usually be all that is needed, although it may happen that morphine will have to be used. If we could employ cocaine with such instantaneous effects about other parts of the body as we can about the eye, we should have at our disposal for general analgesic purposes a drug of far more potency than any of which we can now boast.

Certain acute affections of the ear causing sufficient pain to produce insomnia must, of course, first be treated, so far as is possible, by relief of the particular lesion presented, but if then the insomnia and pain continue, the former will generally disappear with the latter, so that analgesics—best of all, morphine—will be all that is needed. If the condition should, however, become chronic, then some less dangerous pain-reliever should be used. In some irritable and hysterical individuals, however, a very slight irritation of the internal auditory meatus in the form of some little ulceration is often the cause of considerable pain and insomnia, both of which may be relieved by treatment of the cause.

When certain infectious diseases cause pain and secondarily insomnia, it may happen that analgesics alone may relieve, or, as is generally the case, both analgesics and hypnotics will have to be employed.

Intracranial tumor seldom causes insomnia, unless it is syphilitic; indeed, there is often a tendency to too much sleep, verging upon coma rather than insomnia. If, however, insomnia should result in a case presenting symptoms of intracranial lesions, this fact of itself should render us suspicious of intracranial syphilis, and careful trial should therefore be made at once of large doses of the iodide, commencing with thirty drops of the saturated solution of the iodide of potassium, given every four or five hours in a full tumbler of water, Vichy, or Giesshübler, and increasing the dose every day by three to five drops at each dose, until 100 or 200 grains in the twenty-four hours have been reached in cases that have not been treated before for syphilis, and in cases that have been so treated, until a dose shall be reached that is fully double the maximum of what they had ever taken before. I have not much faith in the use of mercury in intracranial tumors of syphilitic origin or in intracranial syphilis. In few of these syphilitic cases will hypnotics or analgesics be found of any use.

The pain of cerebro-meningitis can usually be better treated by ergot, bromide of potassium, and iodide of potassium than by analgesics, and these will often be sufficient to relieve the insomnia if it be dependent upon the pain. Where this is not the case, or where the insomnia is a symptom *per se* of the meningitis, hypnotics must

be used; and of these I think that chloral is usually by all means the best, if the cardiac condition of the patient will warrant it.

The insomnia of febrile conditions will usually require the use of hypnotics, and those used should be governed by the strength of the patient. I prefer sulphonal in these febrile conditions—10 grains of it with an equal quantity of bromide—and, if necessary, a small quantity of opium or morphine, the latter hypodermically. The sulphate of quinine or strychnine can be combined with them with advantage in asthenic conditions. Nevertheless, in none of the febrile conditions should the hypnotics be used, in my opinion, unless the effect of alcoholic stimulants and the bromides has first been tried. Great care should be taken not to render the patient dependent upon any hypnotic.

In neurasthenia insomnia is not apt to be a frequent condition, for I think that most cases of so-called neurasthenia with obstinate insomnia are really simple melancholia, with the diagnostic symptoms to which I have already called attention, and to which I shall again allude. It may happen, however, that neurasthenia will be accompanied by a slight degree of insomnia, and then it should be first determined as to whether the neurasthenia is reflex, lithæmic, aural, or simple. In many of these cases the reflex irritant must be removed, although I may say, in passing, that too much stress should not be laid upon slight conditions of non-nervous organs as possible reflex causes, and the connection between cause and effect should be carefully demonstrated before a positive conclusion is reached. In all these forms of neurasthenia the underlying disease must be treated, and most relief will be obtained from dietetics and rest rather than from hypnotics. Neuralgia and neurasthenia of the simple variety are best treated in the way that I have indicated when speaking of neuralgia. If the insomnia should not be relieved by this general treatment, a glass of milk, a cup of cocoa, or a half-ounce of whiskey in a milk-punch or eggnog, or in water, as the patient may desire, should be given at bedtime, with, possibly, in those who can digest food at this time, a light supper.

In certain exhaustive diseases, such as typhoid fever and anæmia, hypnotics should never be used until they become absolutely necessary, as every purpose will be answered by continued doses of heart-stimulants through the day, with or without quinine, and proper support through the twenty-four hours with alcoholic stimulants, with an extra dose just before bedtime in the shape of a milk-punch or an eggnog; and I have seen hypnotics produce very disastrous results in such cases. If it becomes necessary to use them, a second dose should never be given until the effect of the first one has been observed by the attending physician, and only very moderate quantities should be employed.

I have had occasion to call attention to a certain group of symptoms as diagnostic of simple melancholia, namely, melancholia, obstinate insomnia, and a peculiar sensation to which I have given the name of post-cervical ache, which is located in the head, occiput, or upper cervical spine, and which is differently localized by each

patient. (*Vide* section upon "Melancholia.") By means of these symptoms a positive diagnosis can be made between a commencing melancholia and a simple neurasthenia. The insomnia of this form of disease should never be mistaken for the insomnia from the other causes I have enumerated, and attention to the presence of the post-cervical ache and the peculiar melancholia should prevent any confusion in diagnosis. The treatment of this form of insomnia is the treatment of melancholia. In general paresis hypnotics may be needed, but usually the administration of hyoscine, $\frac{1}{100}$ grain, or hyoscyamine, $\frac{1}{100}$ grain (far preferably the former, because it is much less depressing), together with 10 to 15 grains of the bromide of potassium, will often be sufficient, when given two or three times a day, to quiet the patient and overcome insomnia. Should this not be the case, it will usually be because the excitement has not been removed, and then it will often be wise to make use of sulphonal in 5- to 10-grain doses, two or three times a day, the last dose being given at bedtime, with or without the hyoscine or the bromide of potassium; but this should only be done with robust individuals, or, if temporarily employed with others, the heart should be watched carefully, and in all cases it is usually best to give tonic doses of quinine at the same time. In the acute forms of insanity, such as hallucinatory insanity or acute paranoia, insomnia is not usually more than a temporary symptom, and it can be overcome by any one of the hypnotics. The insomnia of the chronic insanities becomes a feature at certain times of the month or at certain periods of the year, but it is scarcely worth while to interfere with it unless it becomes very marked and disturbs the patient's nutrition, in which case it will be best treated by hypnotics given with a light supper at bedtime, or even with some alcoholic stimulant in debilitated cases.

Certain organic diseases of the cerebrum may produce insomnia, such as tumors, the different forms of meningitis, abscess, and chronic arterial disease; but in these maladies there is more of a tendency to too much sleep, frequently bordering upon coma, rather than to insomnia. In one organic disease of the brain, however—intracranial syphilis—as I have pointed out (*vide* section upon "Syphilis of the Nervous System"), the insomnia in the early stage is apt to be very obstinate, and is accompanied by quasi-periodical headache, the quasi-periodicity being generally nocturnal, although it not infrequently comes in the afternoon or morning, and the headache and insomnia cease abruptly upon the supervention of any hemiplegic or convulsive symptoms. In intracranial syphilis the insomnia is never affected in the least by hypnotics, whilst administration of the iodide of potassium in sufficiently large doses will invariably relieve it; and for this purpose use should be made of the saturated solution of the iodide, beginning with 20 drops after meals in a full tumbler of water or Vichy or Giesshübler, and increasing rapidly by the addition of 5 drops each day to each dose until the insomnia and the headache yield, or iodism has been produced. No arbitrary dose short of these effects should deter one in the administration of the drug. As a matter of fact, it will usually require from 100 to 200 grains daily

for a person who has not been accustomed to much iodide, whilst in old syphilitic cases it will require much larger doses, one in my experience having needed 600 grains in the day. When the iodide is given in these large doses, as I have directed, it is very seldom that any unpleasant symptoms arise from the stomach or the skin. In intracranial syphilis I have little faith in mercury, and I never use it in these insomnic cases unless I am unable to push the iodide far enough, when I endeavor to increase its effect by means of the mercury, although I firmly believe that for some unknown reason iodide alone is much more effective in these cases than mercury alone.

Excluding the insomnia which is caused by the passions, pain, certain exhaustive diseases, simple melancholia, certain chronic insanities, and intracranial syphilis, the most common cause is to be found in the use of certain drugs, stimulant narcotics, or foods; and insomnia from these three latter causes is much more frequent than is generally imagined. We are all familiar with the effect of coffee in producing sleeplessness, and yet there seems to be an idea that coffee can be drunk *ad libitum* in the morning if it be avoided at night. I have found that many individuals have sleeplessness which is produced by this cause. Tobacco in some susceptible individuals will produce insomnia when used in any form, and the only safety for such persons is to eschew it altogether. I am inclined to think that the number of such people is larger than is generally supposed. Tea is also a substance that will produce insomnia, although not in many cases. Some individuals have a susceptibility to certain kinds of wine, so that one individual cannot drink champagne for this reason, another cannot use burgundy, a third cannot employ brandy, and a fourth must abstain from pork.

COMA.

Coma may result from—

Trauma;
Nephritis;
Alcoholism;
Diabetes;
Cerebral hemorrhage;
Epilepsy;
Migraine.

In every case the treatment of coma will materially depend, of course, upon the cause, so that the diagnosis of the latter should be carefully made.

In trauma the patient should be allowed to recover from the shock of the injury, which should be remedied as soon as possible, so that fractures of the bones of the extremities, trunk, skull, or cord may be properly attended to or prevented from complicating matters by pressing upon the viscera which they should shelter.

In nephritis the patient should be purged freely, best by $\frac{1}{4}$-grain doses of a reliable preparation of elaterium, or by croton oil, 1 or 2

drops at a dose, at the same time that a profuse perspiration is induced either by a hypodermic injection of pilocarpine or a full dose of jaborandi.

In the coma of alcoholism there is, of course, nothing to be done but to sustain the patient's strength, if need be, and wait until the effect of the alcohol has passed away.

In diabetic coma free purgation will be most effective.

In the coma of cerebral hemorrhage a careful inquiry should at first be made to ascertain whether it is the cerebral hemorrhage of nephritis or chronic endarteritis, of general parésis or some other structural disease, such as disseminated sclerosis, tumor, etc. The diagnosis of nephritis can always be made by examination of the urine, and possibly by œdema of the eyelids, feet, and hands. The diagnosis of chronic endarteritis can be made by the exclusion of nephritis, by the presence of tortuous and rigid arteries, by a hypertrophied heart, and by the age of the patient. The diagnosis of disseminated sclerosis may be much more difficult unless a history of the patient has been obtained before the coma supervened, although the fact of the patient being a youth or young adult with a tremor may lead to a suspicion of the cause.

CHAPTER XII.

SOMNAMBULISM AND ALLIED DISORDERS.

SOMNAMBULISM.

SOMNAMBULISM is a curious phase of nocturnal cerebration that is most analogous to the condition which is produced by hypnotism, whilst its next closest analogue is the double consciousness which is occasionally observed in epileptics. Although the somnambulistic mind is usually much more automatic than the waking one and capable of a far less degree of reasoning intelligence, there are yet some remarkable instances upon record of the intellectual vigor of this semi-dreaming state. An old friend of mine, a physician of distinction, tells me that when he was at college he had a certain essay to complete, and that he went to bed tired out, intending to complete it the next morning; but when he awoke, he found the essay completely written to the end in his own handwriting with exactly the same vein of logic and subtle thought in which he had begun it, so that he must have gotten up in the night and finished it in a somnambulistic condition. Tuke tells of a schoolboy who had been set to prove the 47th problem of Euclid, Book I., but he was unable at first to master it, and retired to bed still thinking of the subject. He was found by his teacher late at night kneeling on his bed with his face to the wall, pointing from spot to spot as if following a proof in a figure on a blackboard, so absorbed in his occupation that he paid no attention to the light of the candle or when addressed. The next morning the teacher said to him: "Well, John, have you finished your proof?" To which he replied: "Yes, I have; I dreamed it, remembered my dream this morning, got out of bed as soon as I could see, and wrote it out at the window." Another story is told of several ambitious young ladies, who found in the morning that they had learned their lessons without any knowledge of having studied them. This puzzled them all for some time, until late one night the mother, returning home from a visit when the moon was shining brightly, found one of her daughters seated at the window in her nightdress only, sound asleep, with her lesson-book in her hand. In 1878 a man named Frazer killed his child in Glasgow. When he was put upon trial some most remarkable facts were proved as to his somnambulistic feats. He had been accustomed when a boy to fetching milk from a farm near by, so he once got up in the night while asleep, dressed himself, got the pitcher, and went to the farm. At another time, when asleep, he went to a timber yard and moved logs until the rain awoke him. When a young man of twenty-one he was frequently found at the landing-place of a pond, asleep, walking

into the water, loudly calling his sister by name, and reaching out his arms as if to rescue her from drowning. Although the contact with the water sometimes roused him, it did not always do so. He had no recollection whatsoever in the morning of these escapades. At other times he would wake up in the night with a feeling of some great impending evil, such as that the house was on fire, that its walls were about to fall upon him, that his child was falling down a pit, or that a wild beast had got into the room and was about to attack him, so that he would tear his wife and child out of bed to save them, all the time roaring inarticulately; or he would pursue the beast through the room, or he would suddenly seize his companion by the throat with the idea that he was engaged in a struggle with the beast. His eyes would be wide open and staring, he would avoid the furniture, would lift up chairs and throw them at the beast, and had often injured himself. He had frequently attacked his father, wife, half-sister, fellow-lodgers, and a fellow-prisoner in the jail. When he killed his child he had seen "a large white beast flying up from the floor and passing through the back of the bed where the child lay; he grasped at the beast, trying to catch it; succeeded in seizing it, and springing out of the bed, he dashed it on the wall or floor to destroy it." He manifested the greatest horror upon coming to and finding what he had done. A pupil of the school of St. Pons, while somnambulistic, attempted to stab his teacher in the night, was disarmed, and taken to another room, where he slept till morning, when he had not the faintest memory of what had taken place. A certain Madame X., whose story is told by Mesnet, made a suicidal attempt in his presence whilst she was somnambulistic. She made a noose of her apron, fastening one end to a chair and the other to the top of the window. Then she kneeled down in prayer, made the sign of the cross, mounted the stool, put her head in the noose, and tried to hang herself. Mesnet, scientific to the core, very properly let her hang as long as he dared to, and then stopped the performance. At another time she attempted to kill herself by violently throwing herself on the floor, after having failed to fling herself out of the window. At still another time she tried poison, filling a glass with water, putting several coins in it, and hiding it after bidding farewell to her family in writing; the next night, when she was again somnambulistic, she took the glass, but changed her mind again, writing to her family explaining her change of purpose. Mesnet relates some interesting experiments made upon a French sergeant in a condition of somnambulism, demonstrating the excitation of ideas in the mind through the sense of touch in the extremities. This soldier touched a table, passed his hands over it, and then, finding nothing, opened a drawer, took out a pen, found paper and an inkstand, got a chair and wrote a letter to his commanding officer, speaking of his own bravery and asking for a medal. A thick metallic plate was then placed before his eyes so as completely to intercept vision. After a few moments, during which he wrote a few words with a jumbled stroke, he stopped, but without any petulance. The plate was removed, and he went on writing. Water was sub-

stituted for the ink, and when he perceived this, which he did quickly, he stopped, carefully examining the pen but not the inkpot.

The prognosis of somnambulism is usually good, as the phenomena can generally be made to cease by judicious treatment.

The diagnosis is a simple matter, and difficulty concerning it can only come up in a criminal case, when, of course, the previous history of the patient will be of crucial importance.

The physician should endeavor to break up the somnambulistic tendency as well as treat the immediate attack. The general health of the patient should be carefully ascertained, any mental or physical overstrain removed, and any tendency to anæmia should be remedied. The treatment of the immediate attacks should be by the use of belladonna, bromide of potash, circumcision, and removal of genital or rectal irritation, or by suggestion of some device for effecting a sharp tactile impression just prior to the onset of the attack. Belladonna should be used in the form of a reliable fluid extract, one to two or three drops just before going to bed. If this fails, bromide of potash, in water, 20 to 60 grains, according to the age and strength of the patient, should be administered at bedtime. If neither the belladonna nor the bromide of potash acts beneficially, they should be tried in combination, always at bedtime. In some cases suggestion has been of considerable efficacy; thus an epidemic in a school was stopped by peremptory commands to the boys before they went to bed not to have any attacks during the night. In two cases also I was able to bring about a cessation of the attacks by hypnotizing the patient for several successive evenings, and in one case by hypnotizing the patient during the daytime in my office, and then peremptorily making the suggestion that he would have no more attacks. Circumcision has sometimes been found to be of great use, even when there was no genital irritation; and I have known a case in which treatment of a leucorrhœa in a young girl was curative. Ascarides should also be treated, if present. Attacks can sometimes be cut short by a sharp tactile impression, as by placing a tub of cold water in such a way that the patient must step into it in getting out of bed.

ECSTASY, or TRANCE.

Ecstasy is a rapt condition of mind in which ordinary impressions are not received in the ordinary way.

This is a rare condition which generally occurs in hysterical individuals. Probably the most remarkable case on record, and the one which has been best studied, is that of Louise Lateau, of Bois d'Haine. This occurred in 1869, in a village of Belgium, in a girl of twenty-three, whose previous life had offered nothing remarkable. One Friday Louise Lateau noticed that blood was flowing from the left side of her chest, and this recurred every Friday. On each Thursday morning an oval surface, about one inch in length, on the back of each hand, became pink in color and smooth, whilst a similar oval surface on the palm of each hand took on the same hue, and on the upper surface of each foot a pinkish-white square ap-

peared. Examined under a magnifying glass, the epidermis appeared at first without solution of continuity, but delicate. About noon on Thursday a vesicle formed on the pink surfaces, containing clear serum. In the night between Thursday and Friday, usually between midnight and one o'clock, the flow of blood began, the vesicle first rupturing. The amount of blood lost during the so-called *stigmata* varied, and some observers estimated it at about one and three-fourths pints. The blood itself was of a reddish color, inclining to violet, therefore of about the hue of capillary blood, coagulating in the usual way, and the white and red blood-corpuscles being normal both in character and relative proportion. The sanguineous flow ceased on Saturdays. During the flow of the blood the patient was in a rapt, ecstatic condition. The facial expression was one of absorption and far-off contemplation, changing often to melancholy, to terror, to an attitude of prayer or deep contrition. The patient herself stated that at the beginning of the ecstasy she imagined herself surrounded by a brilliant light, then figures passed before her, and the successive scenes of the crucifixion were panoramically progressive. She saw Christ in person, his clothing, his wounds, his crown of thorns, his cross, as well as the Apostles, the holy women, and the assembled Jews. During the ecstasy the circulation of the skin and heart was regular, although at times a sudden flush or pallor overspread the face, according with the play of expression. From mid-day on Thursdays, when she took a frugal meal, until eight o'clock on Saturday mornings, Louise Lateau took no nourishment, not even water, because it was said that she did not feel the want of it, and could not retain anything upon her stomach. During this time the ordinary secretions were suspended. Tuke has resurrected the remarkable case of John Englebrecht, reported by Arnold, of Leicester, in the early part of the century. This man passed into a condition of catalepsy in which he heard everything about him distinctly, but seemed in his imagination to have passed away to another world, this condition coming on with a suddenness which he describes as "far more swiftness than any arrow can fly when discharged from a crossbow." He always lost his sensation from the head downward, and recovered it in an inverse direction.

These conditions of ecstasy or trance are very rare, as I have already said. Much ado has been made about the danger of people being buried alive whilst in this condition, and an indictment was brought two years ago in the city of New York against two well-known physicians, who were alleged (without the slightest evidence) to have made a post-mortem upon a person in this condition. As a matter of fact, there are but very few so-called authentic cases of premature burial, if any. A well-known editor in this city has for many years had the scientific fad of ascertaining the facts about every such supposed case, and in no single instance has he found sufficient evidence to warrant him in a belief that a person had been buried alive.

The prognosis of ecstasy is usually good.

The diagnosis is easily made.

The pathology of the condition is totally unknown.

The treatment should consist of general hygiene, dietetic and medicinal measures, and also of hypnotic suggestion.

CATALEPSY.

Catalepsy is a condition in which there is a peculiar wax-like muscular rigidity, loss of sensation, and complete suspension of normal mental action. The paroxysms last for minutes or hours, and in some cases they even continue for days. The limbs retain any position in which they are placed. There is often a neurotic heredity, and frequently the immediate exciting cause is an emotional disturbance. The prognosis is usually favorable. The pathology is entirely unknown, although in a few cases some cerebral lesions have been reported which were probably merely coincidental. The condition is undoubtedly similar to that which is known as cataplexy (*vide* "Hypnotism") and is, therefore, akin to the hypnotic condition. The diagnosis is made with great ease. The treatment should be by means of general dietetic, hygienic, and medicinal measures, as in somnambulism and ecstasy, and in several instances suggestion has been found to be of great efficacy.

NIGHT-TERRORS.

When a child has night-terrors, as is well known, it starts up in its sleep with a cry, the eyes staring, the face twitching, and seems to be alarmed, whilst no persuasion or soothing has anything more than a slight effect, the child being really in a condition analogous to that of the somnambulist, with the difference that the attack is temporary and does not lead to perambulation. These troubles are very apt to have the same causes as somnambulism, namely, predisposing ones of heredity, bad health, indigestion, constipation, worry, anxiety, and exciting ones of over-eating, intestinal parasites, mental strain, etc. But night-terrors are much more manageable than is somnambulism. They can best be treated by means of bromides, belladonna, warm baths, electricity, removal of any indigestion, and circumcision. When the bromides are given, they should be used in doses of 10 to 30 grains, according to the age of the child, and administered an hour or so before bedtime. The dose of belladonna should be sufficient to cause moderate dilatation of the pupil the next morning and slight dryness of the throat, although it may not be necessary to have these effects continued throughout the day. Occasionally a tepid bath every second night at bedtime will answer the purpose. Electricity is not usually of great value in this malady, although it will occasionally do good. I prefer general faradization, which can best be employed in these cases by having the whole body faradized, with a gentle current just strong enough to be perceptible, by a nurse or mother just before the child goes to bed. For this purpose any good electric battery will answer. It should have two electrodes (see Fig. 71) covered with absorbent cotton, which should

be well soaked in warm water before being used; and these should be kept about two inches apart whilst about fifteen to twenty minutes is taken to go over the surface of the body. Any causes of indigestion in a child with night-terrors should always be removed, and in all cases it is well to have the evening meal a light one. Circumcision is generally of very great avail, for although its effect is only temporary, yet in so functional a disease this is often quite sufficient to lead to a cure. It may even be done with benefit in cases in which there is no phimosis or redundant prepuce.

HYPNOTISM.

Although the phenomena which are now classified under the name of Hypnotism have only been scientifically studied during the latter half of this century, there is yet excellent reason to believe that they were known to the ancient nations of the East of whom there is any history, to the Greeks in their palmy days, to the Romans, to the Egyptians, and to the nations of Europe that slowly evolved into civilization after the downfall of the Roman Empire. The fakirs of India, the magicians of Persia, the oracles of Greece, the seers of Rome, the priests and priestesses of Egypt, the monastic recluses of the Middle Ages, and the ecstatics of the seventeenth and the early part of the eighteenth centuries exhibited many symptoms that were and are still attributed by religious enthusiasts to supernatural agencies, but which are explicable by what we know of hypnotism. The Hesychasts of Mount Athos, who remained motionless for days with their gaze steadily directed to the navel; the Tasko-drugites, who remained statuesque for long periods with their finger applied to the nose; the Jogins, who could hibernate themselves at will; the Dandins of India, who became cataleptoid by twelve thousand repetitions of the sacred word "om;" St. Simeon Stylites, who perched on a lofty pillar, preserved for days an attitude of saint-like withdrawal from earthly things; and even Socrates, of whom it is said that he would stand for hours motionless and wordless—all these were probably instances of auto-hypnotism. But when Mesmer began his career in Paris, in 1778, the attention of scientists was first directed to the subject, although he had written a paper upon the subject some eleven years before and had presented it to the medical faculty of the University of Vienna. Mesmer's career in Paris was a singular one, and it is difficult to form an opinion as to how much he was actuated by true belief and how much by mere sensationalism and cupidity. Certain it is that he was one of the leading figures in the great European capital until the public mind was diverted by the terrible events of the French revolution. His waiting-rooms came to hold such mobs of so-called patients that he finally devised his celebrated *baquet*, which was nothing more than a great tub filled with broken glass and odd remnants of metal, the sides penetrated by pieces of wire, so that the patients could sit around this queer receptacle, and, hypnotizing themselves by grasping hold of the wires, thus economize time! Since the time of Mesmer the

subject has been studied with increasing interest, and the literature of it has grown to be so enormous that Dessoir has lately compiled a bibliography covering some ninety pages of quarto size. Braid, of Manchester, paid great attention to the matter from 1840 to 1860. Of late years Charcot and his scholars in Paris, Pitres in Bordeaux, Ladame in Geneva, Binswanger in Jena, the so-called school of Nancy—consisting chiefly of Bernheim, Beaurnis, Liégeois—Renterghem and Eedem in Holland, in England the Society for Psychical Research and Hack Tuke, have attempted to study the matter in the light of modern methods and clinical knowledge; whilst in America, Grimes, Dodds, Stone, Darling, Durand De-Gros, and Beard, have contributed their quota, and even the quack Perkins made for himself an unenviable reputation with his celebrated metallic tractors. Czermak (1873), Prior (1878), and Hoebel (1876) have demonstrated the existence in animals of similar phenomena, to which they have given the name of cataplexy.

There are many methods of hypnotizing the human being, and those who can be hypnotized, either from natural predisposition or from training, can be put into this condition very readily in different ways that have suggested themselves by experience, either with or without the aid of other persons. The methods are usually as follows: Let the person to be hypnotized take some object in one hand and regard it fixedly; or let the hypnotizer take the hands of the person to be hypnotized, lift them up and move them slowly from above downward, from the top of the head to the stomach, parallel with the front of the body, taking care not to touch the body of the person to be hypnotized; or let the hypnotizer place the person to be hypnotized upon a chair and then look keenly and sharply into his eyes; or let the person to be hypnotized be seated on a chair, then let the hypnotizer say to him, "Think of nothing else but that you will fall asleep;" then in a few minutes let the hypnotizer say, "Now your eyelids are falling; your eyes are becoming more weary; the lids are going nearer together; now they are trembling; now you are beginning to feel tired; now your arms are asleep; now your legs are asleep; now your whole body is becoming heavy and sleepy;" and so on in this vein. Braid hypnotized all his patients by having them look fixedly at an object held or fixed well above the level of the eyes, so that the ocular muscles would become weary, which is the method I generally employ; and he narrates many curious instances of how effectual this was, especially in the case of his man-servant, whom he would usually put into this condition by telling him to watch closely the flame of a spirit-lamp in his laboratory. All these methods can usually be assisted greatly by gentle movements of the hypnotizer's hand across the brow and down the sides of the face, taking care always to make the movements in the same direction, as well as by occasionally closing lightly the eyes of the subject and pressing gently through the eyelids upon the eyeball.

There is a variation in the behavior of hypnotized individuals. Usually, however, the first signs are a closure of the eyes, with a

slightly tremulous movement of the eyelids. Beyond this there are four great manifestations of abnormal nervous and psychical conditions which are grouped under the names of lethargy, catalepsy, suggestion, and somnambulism. In my experience the condition of lethargy is by far the most common. In it the person is like one asleep, and the unconsciousness is so pronounced in some cases as almost to merge into coma. On one occasion, when lecturing upon the subject, I suspected a certain person of malingering, and therefore placed him against the wall when he had passed into a seeming condition of lethargy; but in a few moments, whilst I was proceeding in my lecture with my back turned to him, I heard a thud, and lo and behold! my patient lay prone upon the floor in an alarmingly comatose condition! I have upon a number of occasions seen lethargy as profound as this, although such a pronounced condition is exceptional. In some individuals who have been rendered lethargic there supervenes a curious hyper-excitability of the muscles and the motor nerve-points, so that the limbs can be made rigid by merely passing the hand over them; or the motor nerve-points can be touched with a pencil, and contractions of the corresponding muscles will ensue as if a faradic current had been employed. In others the lethargy can be made to pass into catalepsy, and one of the best means recommended for this purpose by the Parisian school is to open the patient's eyes before a strong light. In some susceptible individuals, so Charcot and his followers claim, the catalepsy may be made to affect only one side of the body by opening only the eye upon that side, and it may be very pronounced. In most individuals who have been passed into the hypnotic condition suggestions may be made of the most curious character, and they will be carried out after the patient is awakened; and at this time also there is sometimes a curious automatism displayed, so that the hypnotized will imitate the hypnotizer's movements or repeat his words. When somnambulism can be evoked, it is by gently rubbing the vertex of the skull of an individual who is already lethargic or cataleptic. Persons in this condition, half conscious, will respond automatically to questions and commands, and they will sometimes display a most remarkable increase of all their mental faculties and sensations. They may be awakened from the lethargy by pressure upon the ocular bulbs, by shaking or by slapping; from somnambulism, by laying a hand upon the vertex; from catalepsy, by putting before them a dazzling light, but not infrequently a stronger peripheral excitation must be employed, such as dashing cold water upon them, blowing into the face, pressure upon the ovarian region, etc. The Nancy school, however, have demonstrated that a simple suggestion or command to the patient to wake up will answer better than any of those means; and this I have verified over and over again. Very many other curious phenomena can be evoked besides those which have been detailed; indeed, it is difficult to say what cannot be done by an individual susceptible to hypnotism.

The so-called cataplexy in animals is a much less complicated phenomenon, as is to be expected from the inferior organization of the nervous system in these subjects when compared with that of the human

being. A chicken, a rabbit, a frog, a crab, a lobster, almost all birds, can be put into a cataplectic condition by simply seizing them firmly and quickly in such a way as to make struggling impossible, and then gently and imperceptibly removing the restraining hand, when the animal will remain motionless in the position in which it has been placed. A chicken can be seized, for instance, put into any position that is desired, held gently but firmly there for a few seconds, and then, if the hands are very gently removed without any abrupt movement that shall constitute a distinct peripheral impression, the chicken will remain motionless for some time, even for a half-hour, as I have frequently demonstrated. Even wild birds of passage can be seized in the same way, and will lie motionless in the hand of the person seizing them when the fingers have thus been carefully withdrawn. During this period of cataplexy birds and chickens keep their eyes wide open, glance around, move their heads and necks in order to do it, and yet remain in the same position. The success of the so-called horse-tamers is probably due to such a condition of cataplexy having been induced. The awakening is done by a slight peripheral impression, such as blowing or touching with the fingers. That the nervous impression is a profound one, although it seems to be so slight, is evidenced by the fact that I have often found frogs, tadpoles, small birds, and even crabs, dead a few hours after they had seemingly recovered from the cataplexy.

It is impossible to doubt that we are dealing with great laws of nervous action in these hypnotic and cataplectic conditions, but it is as yet entirely a matter of speculation as to what the cellular changes are, just as much as it as yet a matter of speculation as to what the cellular changes are in thinking, in sleeping, in the emotions, in neuralgia, in catalepsy, in hysteria, or in most of the insanities. There has been a great war of words waged over the question as to how much of these phenomena are due to hysteria or to suggestion. That the hysterical are more easily hypnotized than others demonstrates that the theory of suggestion cannot be regarded as offering a full explanation, although it probably contains a large portion of the truth. Certainly the cures effected by so-called "Christian science" and "faith cures" are indubitable evidence of the potency of suggestion. The truth of the matter is that the whole subject is one which has as yet been imperfectly studied, and the truly scientific attitude toward it should be neither one of skepticism nor credulity, but simply of expectancy.

Imposture should be readily detected. Almost all individuals who can be hypnotized will pass into the condition of lethargy, and if imposture is suspected, it will be simply necessary to let the lethargy increase, when the facial expression of the patient, the slight pallor, the insensibility to painful impressions, and the appearances of unconsciousness which are familiar to every physician, will show that there is no simulation. The hyper-excitability of the muscles and motor nerve-points in the lethargic condition cannot be simulated, because contracture and muscular movements in the limitation of a given nerve-branch are not within the control of any person's will.

I know of no means, however, of detecting simulation of hypnotic catalepsy or of somnambulism.

The percentage of persons susceptible to hypnotism has never been determined, as so much depends upon the skill and quickness of observation of the hypnotizer, but it is probable that most adults can be hypnotized, although it cannot always be done the first or even the second time, and in some instances it may require a half-hour. Usually the superinduction of this state is not attended by any ill consequences, but some individuals may be so profoundly disturbed by it as to suffer for hours or even days afterward from *malaise*, insomnia, nervous vomiting, nervous excitability, etc.

From a medico-legal point of view hypnotism is a matter of some import, for it is a question as yet not determined as to whether a person cannot be made to commit a crime by suggestion, as was alleged in the trial of the Parisian strangler, Eyraud, when it was claimed that his accomplice in crime, Gabrielle Bompard, had been hypnotized. There can be no doubt that outrage can be effected in the hypnotic condition, and there was a notable trial demonstrating this a few years ago in the south of France, as narrated by Bernheim. Public exhibitions of hypnotism should be forbidden by law, and even private exhibitions should not be held except by competent persons who are acquainted with the proper methods and the dangers of them. It is my rule in my clinic never to hypnotize persons without their full consent, after explaining to them the possible ill consequences.

The therapeutic value of hypnotism is small, it must be confessed. The French authors have done some wonderful things by this method, but it would seem that the Latin race is more hysterical, and from this cause or from some other is more susceptible to hypnotism than the Anglo-Saxon. Moreover, the experiments which have been performed by Charcot have been to a large extent upon individuals congregated within the walls of La Salpêtrière, that great hotbed of neurotics, and some of them have been hysterics of over thirty years' residence in the institution. Certain it is that no such results have been obtained either in England or in this country, except in very rare instances. In hysterical individuals I have sometimes been able by hypnotism to disperse slight functional disturbances, such as pain, mild forms of neuralgia, nervous irritability, even light grades of insomnia. In some cases of dipsomania, or even of intemperance, I have been able to control the extremely nervous condition and the insomnia which is so apt to follow prolonged drinking; but in none of the organic diseases of the brain, or the spinal cord, or the insanities have I ever seen any effect from hypnotism. It is a somewhat dangerous thing, too, for a physician of standing to employ it, for it is associated in the lay mind, and even in the medical, with quackery and sensationalism, and so greatly did it demoralize the discipline of one of my hospital wards that I was reluctantly obliged to forbid it there.

CHAPTER XIII.

DISEASES OF UNKNOWN PATHOLOGY.

FACIAL HEMIATROPHY.

Facial hemiatrophy is a wasting of the bones, subcutaneous tissues and muscles, the latter suffering but slightly. It is a rare disease, and needs only to be distinguished from one-sided muscular atrophy, which can be readily done because in the former there is no loss of electrical reaction in the facial muscles, and because there is no atrophy of the other muscles of the body. It is a chronic affection, usually beginning in early life, increasing slowly for years, and then becoming stationary. It is incurable. The pathology of this disease is not as yet clearly ascertained, but the few autopsies that have been made would seem to show that the lesion is either in the sympathetic, implicating the trophic fibres that run in this nerve, or else that it is a peculiar form of multiple neuritis.

PARALYSIS AGITANS.

Synonyms: Schüttellähmung. Shaking palsy. Parkinson's disease.

History. Paralysis agitans was first described in 1817, by Parkinson, of London. It is a singular tribute to intellectual capacity that nothing essential has been added to his description of the disease, although almost all the well-known writers have considered it.

Clinical History. The onset of the affection may be gradual or sudden, very generally the former. It usually commences in one of the extremities, although occasionally it begins in the lips, as it did in one case of my own, which had a duration of fifty years. The tremor gradually extends throughout the extremities, the muscles of the neck and head, and sometimes the facial muscles and the tongue. This extension is usually bilateral, exceptionally unilateral. The tremor is usually coarse, and in pronounced cases or in mild ones suffering from temporary exacerbation it may consist of a coarse tremor intermingled with movements that almost resemble a mild localized muscular convulsion. The speech soon becomes affected, and grows to be deliberate and solemn, like the ideal speech of a wise man, or that of a slow, hesitating, and deliberate individual. The attitude soon becomes peculiar. The patient holds the head and shoulders stiffly, bending the head and neck slightly forward, and walks with a short, shuffling step. The *facies* is as peculiar as the attitude, and consists of a blank immobility that is very difficult to describe, but that is admirably represented in

Fig. 164. At this stage of the disease the hands are apt to assume the so-called bread-crumbling position; *i. e.*, the thumb and the fingers approximate and move restlessly over one another, as in the act of crumbling bread. There is often a tendency on the patient's part to go forward—so-called propulsion—and this is sometimes so marked that if the patient is once started in a walk forward, his gait becomes more and more rapid, and he cannot stop himself. This is

FIG. 164.

Case of paralysis agitans, showing the attitude, the position of the hands, and the *facies*.

common. In other cases there is a tendency to go backward—so-called retropulsion. This is not often observed. In other cases, again, there is a tendency to go to one side—so-called latero-pulsion—and this is rare. In all the marked cases of paralysis agitans there is a slightly red hue of the face, seemingly indicative of health, but really due to vasomotor paresis. The tremor is generally present only when a voluntary muscular movement is made, and great stress is laid upon this fact as a means of diagnosis by Charcot; but

whilst generally present, it is often absent, especially in the cases that have not arrived at the typical stage, and too much diagnostic importance should not be attached to its presence or absence. Involuntary movements are sometimes present in paralysis agitans. Attempts have been made to derive some diagnostic data by studying the sphymographic tracings of the tremor, more especially to distinguish it from the tremor of multiple sclerosis. Charcot has found that in paralysis agitans the line traced by the tremulous extremity is one of slight tremulous, continuous waves when no voluntary movements are made, but during such these waves are somewhat longer and more irregular. In multiple sclerosis, however, the line is almost a straight one when no voluntary movement is made, whilst in such voluntary movements the line is one of large curves, much larger than at any time in paralysis agitans. Grashey has found the rhythm of the tremor of paralysis agitans in three patients to be 0241, 0190, and 0187. A number of others, among them Marie, Charcot, Gowers, and Peterson, have found the average rate of vibration of the tremor to be from 3.7 to 5 per second. Rigidity of the muscles is almost always present, as is also contracture. The tendon-reflexes are only exceptionally increased. Other symptoms that are occasionally found are: a feeling of excessive heat over the whole body or in certain areas, a feeling of cold or numbness and prickling, pain, increased perspiration, and tachycardia. Most case of paralysis agitans are extremely restless, so that they cannot long remain quiet, and clamor at times, even when almost helpless with the disease, to be put on their feet and allowed to walk about. The mental faculties are usually not at all affected, and I have seen cases of over fifty years' duration, in which the mind, subjected to the most rigid testing, showed no abnormalities in any way. This was notably exemplified a few years ago in the case of a public man of national reputation, who to the last remained the leader of a great party. In some cases, however, there is slight mental dulness or peevishness, and even marked stupidity. The duration of the disease is indefinite, and as it is never cured entirely, it lasts throughout the rest of life, and whether it curtails life is a question that has never been solved. I have seen several cases that lasted thirty years, and two that lasted fifty years, and these all brought the patient up to sixty, sixty-five, and seventy years of age, so it might well be a question whether they would have lived any longer without the disease. But there are exacerbations, followed by remissions, and these exacerbations are often so pronounced as to lead the patient to believe that death is approaching, or the physician to maintain that the last remedy used has caused the remission that follows. In these exacerbations the patient may become, for the time being, perfectly helpless and occasionally semi-comatose.

Etiology. Age is an etiological factor of considerable importance. Most cases occur between forty and sixty years of age, and even up to seventy. Sex appears to be of but little account, although men are somewhat more frequently affected than women.

In some cases, however, the exciting cause may be—

Emotions;
Cold and damp;
Heredity;
Strong peripheral irritation;
Trauma;
Gout;
Typhus;
Hemiplegia.

The emotions are sometimes very distinct causes. One patient of mine was on a ferry-boat crossing the East River, when a baby sprang out of its mother's arms into the river, and, buoyed up by its long clothes, was swept down the strong current until rescued by some 'longshoremen in a boat. My patient, on returning to the cabin after watching this scene, found, to her surprise, that her upper lip was "going," as she expressed it, "like a rabbit's," and this was the beginning of a typical paralysis agitans that culminated fifty years afterward in death. Worry and anxiety have also been noted as causes.

Cold and damp are generally supposed to be the most prominent causes by the European writers, but this has not been my experience, although I have occasionally obtained a history to that effect.

Heredity is a frequent cause, in my experience, although it will often need some patient questioning to obtain a history of it. It may not have been in the immediate parents of the patient, although it may have been running through a family irregularly in different generations. In our New York clinics I have observed an unusual proportion of cases among emigrants from the northern parts of Ireland, or among the immediate descendants within one or two generations of the north of Ireland population, and also to the same extent among the Scotch.

Westphal has reported cases of paralysis agitans following a wound or a laceration, or burns of an extremity.

Trauma is also occasionally observed as a cause, and the same may be said of gout and typhus.

I have seen some five cases of paralysis agitans following a hemiplegia and affecting the hemiplegic limbs, although such cases must not be confounded with those in which the malady affects one limb or one side of the body.

Prognosis. The prognosis, so far as affects life, is generally good, for patients seldom die suddenly of the disease, and, as has been already stated, it is very much a question whether it shortens life. The prognosis of improvement under proper treatment is good also, although I am aware that this is not the opinion of writers generally upon the subject. Some cases, however, will resist all treatment, and these are apt to be those in which the tremor is localized in one limb or on one side of the body, and in such cases the tremulous limbs are apt to become paralyzed and rigidly contractured.

PATHOLOGY. Paralysis agitans has until within a very short time been supposed to be a functional disease of the nervous system, as no pathological lesions were demonstrable. Joffroy claimed in 1872 that he had found the central canal of the cord almost obliterated by pigmentation in three cases, but this condition has been observed in many cases in which paralysis agitans was not clinically manifested; and it was also shown that in the aged are often found such indurations of the membranes as had been claimed as pathognomonic lesions. But this question has again been brought forward by the observations of Redlich, Koller, Borgherini, Schultze, Ketscher, Dana, and Saas, and a new light has been thrown upon it. These authors have shown that there are islets of sclerosis in the medulla oblongata and spinal cord of these cases, evidently having their origin in the vessels. This is a condition which will be known as perivascular sclerosis. Vessels of small calibre are those affected. The intima is slightly proliferated, there is a narrowing of the lumen, and a thickening of the layers of the media, which are also somewhat nuclear, concentrically layered, and hyaline. There is a region of thick, horny tissue outside of these, usually closely approximating it, sometimes with a small intervening space, coloring readily in ammoniacal carmine, and which answers topographically to the adventitial lymph-spaces or to the layers of the adventitia. Hemorrhages or miliary aneurisms have been observed from this cause. There is a thickening of the neuroglia from this point which extends into the surrounding nervous parenchyma, in places surrounding groups of nerve-fibres, and individual nerve-strands only in others. These appearances diminish toward the periphery of the cord. Small islets of sclerosis may become confluent if there are many vessels close together, forming large masses of sclerotic tissue. Where the sclerosis is of small degree and especial groups of nerves are surrounded the nerve-fibres are not affected, but the compression of nerve-fibres is shown where there is a more marked degree of sclerosis, so that diminution in size or entire disappearance of the axis-cylinder and the medullary layer is observed. Even the large vessels outside the spinal cord may also be affected, especially the interfunicular artery and the posterior fissural artery, in the coats of the media. The posterior columns of the cord show these sclerotic changes most clearly, particularly in the middle and ventral portions, in proximity to the posterior commissure. But although the lateral columns are also affected, the lesions are not limited to them, as the Gowers column, the lateral tract proper bordering upon the gray matter, and the direct cerebellar column are implicated in a diffuse manner. Affection of the anterior columns is but very slight, and quite often entirely absent. Thickening of the vessels of the gray matter often occurs, but without any involvement of the surrounding tissue. The cervical enlargement and the lumbar cord constitute the location of greatest implication, the lesions being relatively far less marked in the upper cervical and lower dorsal region, and in the middle and upper dorsal still less so. Implication of the cortical region has also been apparent in some

cases. In some instances streaks of tissue histologically similar to the cortical layer of the cord in this region extended into the lateral columns, particularly in the region of the posterior portions of the lateral columns, disappearing around the nerve-fibres; and a diffuse sclerosis of slight degree, with increase of the septa and of the neuroglia, evidently not of vascular origin, appeared in other cases, and Redlich found in one case a diffuse sclerosis of the perivascular character which has been described. The ganglion-cells of the anterior horns were markedly pigmented in almost all cases, the whole body of the cell being sometimes filled with yellow granules concealing the nucleus, and leaving only a small portion of the protoplasm visible, this process, however, being unusual, the form and processes of the cell generally remaining. Clark's columns also present a high degree of pigmentation. Proliferated ependymal cells usually obliterated the central canal. A large number of amyloid bodies are found. Slight thickening and great pigmentation of the pia of the cord were also usually present. The anterior and posterior roots were unchanged, although the vessels have been found widened, but not thickened. Lissauer's and Clark's columns are usually found to have preserved their nerve-fibres. The axis-cylinders had a varicose shape in one of Dubeuf's cases, and the upper and cervical dorsal cord was found to be the main site of the lesions. Aneurismal enlargements were observed in a case of Borgherini's, with a slight affection of the posterior columns, whilst he discovered marked alterations in the gray substance, in which he differs from most authors. In the medulla oblongata Ketscher and Dana have found similar alterations. Similar lesions of the vessels have been found in the cerebrum in some instances, the sclerosis, however, being absent. Alterations have also been described in the peripheral nerves and muscles by Saas, Redlich, Borgherini, and Ketscher, these changes consisting of proliferated connective tissue and atrophy of the muscular and nerve tissue, with thickening of the vessels.

It has been a matter of discussion as to whether senility brings about naturally these changes that are claimed as characteristic of paralysis agitans. The appearances are fairly constant in about twenty cases which have been examined so far, and in most of them the clinical phenomena and the severity of the pathological lesions bear a fair ratio to each other. But it is a well-known fact that in aged people who have presented no symptoms of paralysis agitans slight thickening of the membranes, obliteration of the central canal of the cord by proliferation of the ependymal cells, increase of amyloid bodies, and pigmentation of the ganglion-cells are frequently found. The observations of Redlich, Leyden, and Demange have shown this. Most authors are inclined to regard the alterations in the vessels, the increase in connective tissue running in from the peripheral tissue of the cord, and the perivascular sclerosis as purely senile, although neurological pathologists differ in this opinion. In a large number of observations made upon old people by Ketscher similar alterations of the vessels to those which have been described in paralysis agitans were found, although in less

degree, and the outer horny layer, as well as the perivascular sclerosis, is usually wanting, and it is also noted that paralysis agitans may occur in young adults, although it generally occurs in individuals toward fifty. Demange has described a progressive tabetic sclerosis of the lateral and posterior columns, of vascular origin, characterized by marked contractures, first of the limbs and subsequently of the arms, without sensory defect, sensory irritation, or decided paralysis, with primary increase of the tendon-reflexes, followed by loss of them. These cases, according to this author, are those of chronic myelitis, caused by an endo- and periarteritis, of the general character of an atheroma. Similar alterations in the aged, attended with muscular enfeeblement, have been described by Copin, although they lack the contracture described by Demange. The senile paraplegia of Gowers will probably rank with this class of cases. A case has been described by Buzzard of a man of thirty-nine years of age having syphilis, then symptoms of diplopia, entire disuse of limbs in walking or standing, although movements of the limbs while in bed were made freely, patellar reflex wanting, one-sided atrophy of the optic nerve, immobility of the pupils, and marked sensory disturbances in the lower extremities, and in whom was found endo- and periarteritis to a considerable extent, with connected sclerosis in the lumbar and cervical cord, in the posterior columns especially, and also in the lateral columns, although in a much slighter degree. Histories of several cases are given by Redlich in which aged people manifested an alteration of the vessels with perivascular sclerosis, particularly in the ventral portions of the posterior columns, the cervical cord being but slightly affected, while the dorsal and lumbar cord were considerably altered, and in the columns of Goll a slight alteration was observed, consisting of a small degree of ascending degeneration, although but slight involvement of the lateral columns was discovered. It is apparent from all this that the localization of the lesion in paralysis agitans is more pathognomonic than its character, for there is no reason why in the cases of Gowers and Demange the symptoms of paralysis agitans should not have been produced by the same lesions if they had been located differently. Tremor and contracture constitute the two salient symptoms in paralysis agitans. It is suggested by Blocq that an affection of the muscles may be the cause of the contracture, but this has not been demonstrated, and we must therefore attribute to the spinal lesion both the tremor and the contracture. Were it not for the fact that a large proportion of the autopsies have shown that the posterior columns are mainly affected, it would be easy, with our existing knowledge, to suppose that both the contracture and the tremor could be accounted for by the anatomical location of the lesion in the lateral and anterior pyramidal strands. It would therefore seem as if the implication of certain fibres in these posterior columns must, under certain circumstances, be held accountable for the clinical manifestations. The only explanation, however, for the fact that the implication of these posterior columns did not produce the symptoms of paralysis agitans in the cases of Buzzard, Gowers, and Demange is the supposition that a different set of fibres is affected in each of the cases.

In the cases of Redlich and Demange there was no tremor and only a slight contracture, so that by exclusion it would seem probable that the tremor was probably caused by affection of the lateral columns and was not due to implication of the posterior columns. It seems probable, however, that implication of the posterior columns may cause contracture by irritation of the anterior ganglion-cells through the sensory-motor tract of Cajal, described on p. 32.

Diagnosis. The diagnosis of paralysis agitans is easily made in the majority of cases. The age between forty and sixty, the coarse tremor, the characteristic attitude, the bread-crumbling position of the fingers; the slow, deliberate, high-pitched speech; the peculiar *facies*, the tendency to ruddiness of the complexion—all are symptoms which are readily recognized in patients, and by which the diagnosis of the disease can often be made on a ferry-boat or in a car. Non-typical cases are very difficult to diagnose. Thus, there are patients in whom the tremor is absent, and here the diagnosis must be made by the other symptoms. Again, there are patients in whom the tremor is present, but who do not present the characteristic speech, attitude, facies, or color, and if this occurs in a patient near thirty, it may be absolutely impossible to make the diagnosis without the aid of sufficient time to observe the progress of the malady. The character of the tremor is no longer thought to be of the importance which it was supposed to have. In the main, I do believe, the tremor of paralysis agitans is present when the patient is at rest or performing voluntary movements; but there are many cases, especially in the early stages of the disease, in which the tremor is only present upon voluntary movements, or is greatly exaggerated at such times. The sphygmographic tracings, of which so much has been made, are open to exactly the same objection, because, whilst the tremor is generally continuous and fine, there are many cases in which it is not continuous, and in which it is at times very coarse. The diseases from which paralysis agitans should be differentiated are—

Disseminated sclerosis;
The tremor of lead, copper, and mercury;
Alcoholism;
Chorea.

The typical case of disseminated sclerosis has a tremor that is present only upon voluntary movements—the so-called intention-tremor—and there are also nystagmus, an interrupted, scanning speech, the patient is a young adult or a child, there may be symptoms of focal disease of the brain superadded; and the characteristic attitude, facies, bread-crumbling position of the fingers, slow, deliberate speech, and ruddy color of paralysis agitans are all lacking.

The tremor of lead, copper, and mercury is usually a fine tremor, and in lead-poisoning other symptoms of the disease will be present, such as the "lead-line" and the wrist-drop, whilst the history of the use of lead may be obtained; and in tremor from mercurial or copper-poisoning the history of the use of mercury may be obtained in the one case, whilst there will usually have been some history of

ptyalism or the mercurial cachexia, and, on the other hand, in copper-poisoning the patient will have been engaged in using copper in some toxæmic way.

In alcoholism, especially in the acute form after a debauch, there is often a fine tremor, and this may sometimes be of the intention-tremor type; but the history of the patient and his general aspect will easily make the diagnosis clear. In some cases of chronic alcoholism there may also be a tremor, but this need only be referred to in order to put the observer upon the alert, for the diagnosis can readily be made by the history of the case and the well-known symptoms.

In ordinary chorea of the Sydenham type there should never be any mistake in making a diagnosis, because the muscular movements are fibrillary in character, either quickly beginning and quickly ending, or more gradual in their beginning and ending when they are of the athetoid variety, and the patient is almost invariably a child. In some cases of chorea, however, that have become chronic, generally from lack of proper treatment or of the original attack of the relapses, the tremor may become so marked and so intermingled with muscular spasm as to lead the inexperienced observer to confound the disease with paralysis agitans; but a history of the case and the fact that the movements are of the type that have already been described will be sufficient to make the differential diagnosis. It is unfortunate that some writers have designated as choreic tremor the fine jerky fibrillary movements common to chorea.

Treatment. The treatment of paralysis agitans should be definite and energetic. All the drugs that have been vaunted as specifics are wellnigh useless except for temporary effect. The patient should be restricted in his expenditure of energy; in other words, the tremulous muscles should be put in a position of comparative rest; and this can best be done by keeping the patient in bed up to noonday for several weeks at the outset of treatment, and at the same time prohibiting walking or fatiguing drives. As the patient grows better he may be allowed to get up at the usual time, but he should religiously avoid fatigue, and if any error is made it should be on the side of too much rest rather than too little. This part of the treatment should be carefully studied, and the most minute directions should be given about it by the physician. It will not do to advance the general principle, but the patient must be told just how many hours he should rest, just how much exercise he should take, just what time he should go to bed, etc. Without this restriction of the expenditure of energy no results will ever be obtained except temporarily. The diet of the patient should be varied, abundant, and nourishing. It should at least consist of three good meals a day, and generally it will be advisable to have at least a quart of milk taken in addition to this in the twenty-four hours, and in some cases it may even be useful to advise that a pound of beef made up into beef-tea shall be taken in the twenty-four hours, either in the form of home-made beef-tea or some one of the beef-extracts or peptonoids. Alcoholic stimulation should be continuously employed, two or

three ounces of whiskey being given in the twenty-four hours, or a bottle or two of a strong ale, or a fairly equivalent quantity of alcohol in the shape of some of the wines. Malt-extracts are also of great value in aiding this process of nutrition. In prescribing them it should be remembered that diastase is that portion of the malt-extract which is alone of value to the patient, and it is therefore of no consequence what particular preparation of malt-extract we employ, provided only that it is fresh and reliable. The liquid malt-extracts with alcohol had better not be employed unless they are more pleasant to the patient than the solid malt-extracts, inasmuch as the alcohol can be obtained in a more precise way in the manner that has just been indicated. Tonics should always be prescribed, and the best of these is the sulphate of quinine, 2 or 3 grains three times a day, given an hour or two after meals to avoid any disturbance in digestion, best in the form of the tablet triturate or in capsules, and never in solution with an acid, which is apt to upset the digestion. In addition to these measures of rest, diet, alcoholic stimulants, and diastase, remedies should always be used directly to control the tremor. The best of these is hyoscyamine, the crystallized and never the uncrystallized form, as the latter is more prone to produce unpleasant constitutional effects. The dose should be $\frac{1}{100}$ grain once or twice a day, continuously used for weeks or months as may be necessary. If the hyoscyamine alone does not control the tremor sufficiently, 10 grains of the bromide of potash may be given with it once or twice a day, usually also for months. Hyoscine, either the hydrochlorate or the hydrobromate, is also a very useful drug, as it can be given much oftener and has none of the unpleasant effect of the hyoscyamine in dilatating the pupil and causing dryness of the mouth. This treatment by rest, alcoholic stimulation, diastase, and muscular sedatives like hyoscyamine and bromide, is the treatment of paralysis agitans—not one element of it alone, but all. The ruddy color of the face so often observed has, I think, misled observers into unconsciously believing that the disease was not in need of stimulant treatment, and therefore has led to the exclusive use of the sedatives, as the result of which patients have been unduly depressed. In reality, however, the ruddy flush of the face is a vascular paresis, and I have found that the stimulating and nourishing treatment alone will do more than the use of the sedatives alone. Galvanism is almost invariably of great value, and it seemingly acts as a nervous stimulant of high order. It should be administered at least three times a week, with one large electrode (Fig. 67) placed on the nape of the-neck, and another of Fig. 68 opposite the lower dorsal spine. The skin should be wet with hot water, and the electrode should be thoroughly moistened with the same. A current of 3 to 5 milliampères should be passed for three to five minutes, and, as the patient becomes accustomed to the current, this may often be advantageously increased to 10 to 15 milliampères and to a sitting of five to fifteen or even twenty minutes. With this treatment I positively assert that most cases of paralysis agitans can be controlled, and the life of the patient made comparatively pleasant. After

successful treatment by these means for a period of eight to twelve weeks, the galvanism may be discontinued, the hyoscyamine and the bromide gradually withdrawn, and the patient allowed simply to keep up the rest that has been found advisable, as well as the diet, the alcoholic stimulant, and the diastase. Usually months or weeks will intervene before another exacerbation occurs, and this can be controlled by the same means. I am accustomed to explain all this to my patients, so that they will expect another exacerbation at some time, when they need not feel discouraged and fear that the disease is becoming uncontrollable. I tell them frankly that they cannot be cured, but that they can be materially helped. Unfortunately, however, the cases of paralysis agitans which occur after hemiplegia, affecting only one limb or one side, cannot be treated with much hope of relief; but a sharp distinction should be made between these cases of paralysis agitans occurring after hemiplegia and the cases of sudden onset in one limb, for the latter are usually only the localized onset of a general paralysis agitans which is subject to the therapeutic laws that have just been detailed.

ASTASIA-ABASIA.

Jaccoud first described this disease, in 1864, as "ataxia with defect of automatic co-ordination." Weir Mitchell wrote of it, in 1881, as "hysterical motor ataxia." In 1883 Charcot and Richer called attention to it as "a special form of motor impotency of the lower limbs, with defect of co-ordination relative to standing and walking." Numerous other authors reported cases, and, in 1888, Blocq collected eleven, and gave to the disease the name that it now bears. Since then there have been numerous articles.

The disease consists of an inability to stand erect or walk normally, although there is no impairment of sensation, of muscular strength, or of the co-ordination of other movements than walking or standing of the lower extremities. In attempting to walk, the legs become spasmodic. There are rapid flexions and extensions of the legs on the thighs and of the thighs on the pelvis. The steps are short and the feet drag; or the body may make great oscillations if the patient stands, walks, or sits, and the head and arms make rhythmical movements; or walking may become impossible, and the patient leaps on to one foot and then on to the other, the body and head oscillating as he advances; or he can walk cross-legged, or by raising the legs high; or he can walk on his hands and feet; or he can walk at certain times and not at others; or he can hop with both feet together, or walk with great strides with the arms extended; or he may be able to use his legs perfectly, if suspended. If the attempt to walk is persisted in, all these phenomena become intensified, and the patient may fall. The most common form of these varieties is the paralytic, wherein the legs bend under the patient in the attempt to walk. There is no rigidity, spasm, or incoordination, and the muscular strength is good whilst the patient is in bed, is sitting, or is suspended. Almost as frequent as the

paralytic form is some variety of spasm or ataxia, the most common being that wherein walking is embarrassed by erratic movements and stiffening of the legs, causing a sort of tremor, like that of spastic paraplegia. Less frequently there are sudden flexions of the legs, so that the equilibrium of the body is nearly overthrown, at the same time that there are exaggerated and sudden flexions of the arms. Charcot has called this type the choreic variety ; and in some cases it has been associated with so-called epidemic chorea, although it must be borne in mind that this so-called epidemic chorea is probably hysteria. A rare form is a saltatory variety. Any emotional disturbance increases the difficulty of walking. A tendency to go backward, or retropulsion, has been observed, as has also a tendency to go forward, or propulsion. A curious phenomenon in this case is that the patients can use the legs perfectly well while they lie in bed.

Astasia-abasia has often a distinct history of heredity. It occurs in about equal proportions in the two sexes. Most of the cases are associated with hysteria, but it has also been seen in conjunction with chorea, epilepsy, such morbid impulses as agoraphobia, etc., as well as with dementia, confusional insanity, exophthalmic goitre, and neurasthenia. Whether or not it is always an hysterical symptom has not yet been determined ; but Blocq has hypnotized a patient, and made her the suggestion that she no longer knew how to walk, and thus produced astasia-abasia ; and when he furthermore suggested to her that she could not walk, paralytic abasia ensued. The cases which I have seen have certainly impressed me as being hysterical, and Blocq's experiment would seem to be conclusive. But this malady has been associated with many diseases which are certainly not hysterical, and the question has yet to be determined whether astasia-abasia can be a symptom of these diseases, or whether it indicates the association of hysteria with them.

The prognosis of astasia-abasia is good. Children recover, as a rule; but relapses occur in adults, and exceptionally these are frequent. It is never fatal.

The treatment is that usual in cases of hysteria (see "Hysteria"). Knapp suggests that a distinctly volitional act be substituted for an automatic act, and hence in his case he suggested the balance-step with remarkable success.

CHAPTER XIV.

REFLEX NERVOUS DISORDERS.

TIME was, and not more than a decade ago, when it was very much the fashion to ascribe almost all nervous disorders to reflex from non-nervous viscera, and cases of what are now well recognized as cerebral palsies of childhood, amyotrophic lateral sclerosis, myelitis of the anterior cornua, pseudo-muscular hypertrophy, myotonia, epilepsy, chorea, and a host of other nervous affections were anxiously examined for phimosis, strictures, dyspepsia, swallowed pins, constipation, uterine or ovarian symptoms, and so on and so forth; and if any of these non-nervous causes were found, the mental symptoms were supposed to be thoroughly explained. In reality this belief is a kind of atavism, for Hippocrates, who lived 460 years before Christ, believed that the causes of insanity lay in the four cardinal fluids: the blood, mucus, the black and yellow gall; and it was not until 160 years after Christ that Galen made the distinction that many practitioners have not yet made, namely, that there were primary insanities due to disease of the cerebrum, and secondary or reflex ones due to an affection of non-nervous organs. Within the last decade this doctrine of reflex nervous disease has been carried to such an extreme that there are medical men of standing who would have us believe that almost all mental and nervous maladies can be cured by the relief of that form of ocular asthenopia which is due to insufficiencies of the ocular muscles, and not long ago I had a surgeon tell me that he could abort a pneumonia by cutting a deep stricture. Neurologists, however, have grown to be very skeptical in regard to the frequency of reflex nervous disorders. Every educated medical man knows perfectly well, of course, that a malady of the abdominal, thoracic, and pelvic organs may produce general symptoms of a nervous character, as well as fever, insomnia, restlessness, jactitation, and vomiting, and every practitioner of experience knows also that slight lesions of non-nervous organs may reflexly produce nervous symptoms; but every neurologist of experience knows, in addition, that almost every nervous malady, of which I have just given a partial list, has at first been thought to be a reflex disorder, and that with the advance of neurological knowledge disease after disease has been removed from this category. The questions, therefore, that neurologists have asked themselves of late have been, first, What is the frequency of reflex nervous disorders? second, What are their characteristic symptoms? and third, With what non-nervous maladies are they most prone to occur?

First, as to the frequency of reflex nervous disorders. It may be positively stated that they are extremely rare, and I have no doubt

whatever that every neurologist has had an experience identical with my own, namely, that many and many a case of so-called reflex nervous disorder will be resolved into a familiar nervous disease which has not been recognized. Statistics are perfectly valueless upon this question, because everything depends upon the experience and the knowledge of the person making the diagnosis, and it is a significant fact that none of the well-known neurologists of the world have reported cases of reflex nervous disorders within the last ten or fifteen years.

Second, as to the characteristic symptoms of reflex nervous disorders. To this it may be answered that the very essence of a reflex nervous symptom is its caprice, so to speak, so that we cannot speak of its characteristics. Thus, it has been said that uterine disorders are prone to produce a peculiar, distressing, aching sensation at the vertex or in the dorsal spine; that insufficiency of ocular muscles causes a peculiar brow-ache; that indigestion induces an aching sensation over the frontal region, and that hepatic trouble causes vertigo. But these different sensations and symptoms are seen so frequently with other disorders than those with which they have been correlated that they are but a broken staff to lean upon in diagnosis. The truth is that when a man presents nervous symptoms, the first question is as to whether he has a nervous malady, the determination of which question involves an accurate and extensive knowledge of nervous and mental diseases; and it is only when all these nervous and mental diseases have been positively excluded that a nervous reflex can be suspected, and this can only be absolutely diagnosed when it is positively found, and time or medical experience has shown that it is the cause of the nervous symptoms.

As to the question which non-nervous maladies are most prone to produce reflex nervous symptoms, it may be said that the following diseases should be considered in a case of doubtful diagnosis, namely—

Nephritis;
Pelvic disorder in the female;
Disease of the male urethra or seminal vesicles;
Intestinal disorders;
Lithæmia;
Errors of refraction;
Insufficiency of ocular muscles;
Aural disease;
Anæmia and leucocythæmia.

In some cases of nephritis the nervous symptoms may be the ones mainly to attract attention, and this is especially apt to be the case when there is a cirrhotic kidney; but a careful examination of the urine, and sometimes an examination with the ophthalmoscope or the sphygmograph, will make the diagnosis perfectly plain. In my experience the general practitioner is much more prone to make a mistake in this matter than the specialist, contrary to the general opinion, for scarcely a month of my life passes in which a case does not come to me of this kind. I had not long ago under my care a

lady who had been treated for two years for supposed neurasthenia, and yet who was found to have albumin in the urine to the extent of 1 : 20; whilst a young man once came to me who had been under treatment for over three years for supposed hysteria, in whom I found a typical albuminuric retinitis, such as represented on page 158, and whose urine showed a large amount of albumin, with numerous hyaline and granular casts. In all suspected cases of this kind a most careful examination should be made, not only by the ordinary test of nitric acid and heat, but also by the more modern tests, and there should be a microscopical search for casts. Care should also be taken to have a specimen of the urine for twenty-four hours, and the whole quantity passed during that period of time should be carefully measured; and, if necessary, repeated examinations, quantitative and qualitative and microscopical, should be made. If these precautions are taken, there should never be a mistake in diagnosis

Pelvic disorder in the female is popularly supposed to be the cause of many reflex symptoms, but I do not think that lesions in this locality are any more prone to act reflexly than lesions of other non-nervous organs except so far as they act psychically, because every woman is only too prone to believe that every uneasy feeling is due to a uterine disorder, and when this opinion is confirmed by some physician it adds a certain satisfaction to her mental torture. Gynecological surgery is so fascinating, the temptation to report a series of 100 cases is so great, and women are such easy victims, that an enormous affirmative literature has grown up upon this subject, to be met by an equally enormous negative literature on the part of neurologists, as an outcome of which the brain and spinal cord have been almost transferred from the skull and the vertebral canal to the pelvis, and anatomical teachings have been wellnigh revolutionized in the mind of the general practitioner. Not long ago I showed to my class a woman who had had for many years attacks of obstinate vomiting, which had resisted every therapeutic device. She came under the care of a well-known gynecologist, who performed a double ovariotomy. For several weeks after this she was regarded as cured, but gradually the old vomiting returned, when the gynecological surgeon opened the wound, thinking that there might be some inflammatory adhesions, but found nothing abnormal. After this exploratory operation the patient was again relieved for several weeks, when the vomiting again returned. She was then sent to me by the gynecologist with the frank statement from him that he had never regarded the ovariotomy as advisable, but had only done it at the request of a well-known teacher of general medicine. Upon examination I found that the woman had been a life-long hysteric, had inherited hysteria, had had hysterical convulsions, and had the characteristic hysterical concentration of the field of vision, with patches of anæsthesia in the upper extremities. She stated that the vomiting had been so violent for weeks past that she had been obliged to live on a pint of milk and a few pieces of bread in the twenty-four hours. As she was fairly plump and well nourished, I doubted her statement, and directed one of my assistants to follow her when she

left the clinic, in order to see where she went and what she did. To our great delight she at once directed her steps to a peanut-stand, bought a pint of the luscious fruit, and meandered her way merrily home, strewing her track as she went with peanut-shells. So when the fair patient again visited my clinic I told her what we had discovered, at which she flew into a violent temper, anathematized us, and has not since done us the honor of seeking our professional advice. I state this, contrary to the rule which I have followed in this book of interpolating no histories of patients, because the importance of the subject warrants me, I think, in illustrating by a case what I so frequently observe, namely, the overweening and illogical anatomical and unphysiological tendency to regard as a pelvic reflex in the female what is really a familiar nervous malady.

Until within a very short time it has been regarded as legitimate surgery to perform an ovariotomy upon an epileptic female, but there is not a case upon record in which epilepsy has been cured by such an operation or by any operation upon the female pelvic organs, although temporary effects may be obtained by such operations, as they are obtained by many others. (See "Epilepsy.") It may be positively stated that no nervous disease is produced by pelvic diseases in the female, although there may be general nervous symptoms caused in this manner. Nevertheless, I believe that an operation upon the pelvic organs of the female is often a very useful therapeutic measure in certain cases of nervous disease. This may seem like a paradox, but it is very far from being such. Opium will relieve pain, yet a man who would get up and say that opium is the cause of all pain would simply excite the merriment of nations; and in the same way the fact that all the component factors that make up a surgical operation upon the female pelvic organs act as a curative or palliative measure does not prove that it causes them. Incidental to the operation is the mental expectancy aroused by the universal belief of women that pelvic disorders in them are capable of producing almost anything, the profound physical shock of the etherization or chloroformization, the bloodletting, the low diet, the rest after the operation, often for months, and frequently the relief of some pelvic source of discomfort; and all these causes, as a whole, produce an effect that may be very potent in certain nervous and mental disorders. I therefore frequently advise an operation, sometimes where there is no local disorder, in cases of mental disease that are very tardy in their convalescence and in which I desire to produce a profound mental and physical effect. On the other hand, it must be remembered that cases of mental disease have been reported as immediately following operations upon the female pelvic organs, and I have seen fifteen such cases myself; so that this is a danger not to be disregarded, and is one which has caused me considerable perplexity. My experience, however, has led me to believe that it is safe to operate with the end in view that I have stated when the pelvic lesion is a slight one, or when the patient is robust in general health, or when there has been some slight pelvic lesion that has caused the patient much anxiety, or when there is no local lesion and the

patient's general health is robust; whilst it is dangerous to operate on patients who are very nervous and apprehensive, or when the pelvic lesion is very serious, or when the general health has been greatly depressed, or when the patient is averse to the operation. In accordance with these rules of my own I have frequently had my gynecological friends curette the uterus, remove a polypus, treat an abrasion of the os, and do similar operations, and I have almost invariably had good results follow. Not long ago, for example, a patient was under my care for hallucinatory insanity, which had lasted upward of eighteen months, from which she had recovered so far as to be relieved of her hallucinations, and to be comparatively docile and reasonable in her behavior, although she was still moody, occasionally depressed, and at times eccentric in action and thought. Medicines had lost their power, and she drifted along week after week in this listless, discouraging way. I advised a gynecological friend whose patient she was to curette the uterus, although he was exceedingly reluctant to do it, and dubious as to its effect. The patient, however, was not only willing, but very eager to have the operation done. The success was brilliant, and in the course of six weeks she went home, I am told, completely cured, and has had no return of her malady in the five years that have since elapsed. I do not believe, however, that ovariotomy is ever warrantable in these cases. It has also been my fortune to meet with a number of cases with ovarian tenderness, frequently bilateral, without hysterical symptoms, occasionally occurring in paroxysms lasting for a few days, a week or more; as well as cases of ovarian disease, which is often suspected, not because there are positive evidences to warrant such a diagnosis, but rather because of the localization of the symptoms; and in several of these cases I have seen cures effected by galvanization with large electrodes (Fig. 68), one being applied over each ovarian region and a current of 10, 20, or 30 milliampères being passed for five or ten minutes every second day for a period of two to three weeks. The objection will be made by gynecologists, I am well aware, that I am not a gynecologist, and may therefore have overlooked some organic local disease. It is therefore proper that I should state that all of these cases of mine have been examined by distinguished gynecologists, and that my attention was first called to the matter by a case which I took to a gynecologist for him to operate upon, when, very much to my surprise, he refused to operate, had nothing to advise, and left me to my own devices, so that, knowing of nothing else to do, I experimented with galvanism, and, greatly to my relief, cured the patient. These cases have, therefore, seemed to me to be local neuralgia of a peculiar type, often made worse by the menstrual period, and frequently following some predisposing cause such as malaria, lithæmia, or depression of the general health.

Diseases of the male urethra have been supposed to play as large a part in the causation of reflex disorders as the pelvic lesions of the female. I believe, however, that the one belief is as much due to defective observation as the other. It is undoubtedly true that

nocturnal enuresis in children and childish disorders akin to somnambulism and night-terrors, as well as these two latter affections themselves, can sometimes be relieved by circumcision. It is also true that strictures of the male urethra and affections of the seminal vesicles may cause reflex pain and uneasiness in the nerves coming off from the lumbar and dorsal cord. But nocturnal enuresis, somnambulism, and night-terrors can be relieved often much more effectively by other measures than circumcision, and the reflex pain and disorder due to stricture and to disease of the seminal vesicles are generally made worse in my experience by any treatment addressed to the urethra and the seminal vesicles that does more than relieve the stricture causing obstruction to the passage of urine, or the inflammation and occlusion of the seminal ducts that prevent the passage of semen. In the chapter upon "Somnambulism," "Night-terrors," etc., many remedies are spoken of that are quite as effective as circumcision, and much less unpleasant. In the adult male afflicted with disease of the seminal vesicles, or a stricture, and who has reflex nervous symptoms, there will often be found a large degree of hypochondria which cannot be relieved by any treatment addressed locally to the urethra or the vesicles. That circumcision will relieve all the varied organic nervous diseases which it was claimed to relieve, and that it will cure epilepsy and chorea, have been disproved time and time again.

Intestinal disorders will sometimes cause vertigo, slight insomnia, and general nervousness, but this very rarely; indeed, the rule is that intestinal disorders do not produce general symptoms, and when they do, they are so pronounced as to make the diagnosis an easy one. In the section upon "Neurasthenia" I have stated my doubt as to whether the cause of the malady might not be some error of digestion in the stomach or the intestine, but this is so largely a matter of uncertainty as yet that it is impossible to speak positively either one way or the other. Epilepsy has seemed to me in some rare cases, as stated elsewhere (*vide* "Epilepsy"), to be excited in a predisposed individual by intestinal disorder, and Herter has described some experiments to show that toxic substances capable of causing epilepsy may be generated in the intestines. Nevertheless, this cause of epilepsy is infrequent and not general, as has been popularly supposed, and even in these rare cases it must be carefully borne in mind that it is an exciting cause and not a predisposing one.

The relation of lithæmia to reflex nervous disorder has been thoroughly described in "Neurasthenia."

Errors of refraction are sometimes a cause of vague nervous disorders, such as slight dizziness, general nervousness, inability to concentrate the mind, etc. But this is a rare cause, and even Mitchell, who has called attention to the subject, has recently so stated.

What has been said of errors of refraction is also true of insufficiencies of ocular muscles causing so-called ocular asthenopia. In cases of migraine in which the attacks have become frequent and violent, a relief of muscular insufficiencies will often act remarkably well as a palliative; and this is also the case in certain vague ner-

vous symptoms in neurotic individuals. There has never been the slightest proof adduced, however, that relief of muscular insufficiency will cure chorea, epilepsy, any of the insanities, or neurasthenia, or even that it will act as a palliative in these cases, and I have derived the materials for this statement from many cases under my own observation.

Aural disease as a cause of reflex nervous disorder is thoroughly discussed in the next chapter, upon "Ménière's Disease," and I can add nothing to that.

Anæmia and leucocythæmia will sometimes cause pronounced headache and neurasthenia; seldom, however, vertigo, and never convulsions. In certain cases of insanity it has been demonstrated that anæmia is marked, but it is extremely probable that the anæmia is much more frequently an effect than a cause in such cases.

The diagnosis of a reflex nervous disorder, therefore, will be by exclusion of all non-nervous and mental diseases capable of producing similar symptoms, and then by the examination of the different organs which we have been discussing. The treatment will vary with the cause.

CHAPTER XV.

MÉNIÈRE'S DISEASE.

In 1861 Ménière observed a case with intense vertigo which died, and in which he found certain hyperæmic and hemorrhagic appearances in the labyrinthine structure of the internal ear, and since then the name of Ménière's disease has been given to vertigo occurring with lesion of the internal ear. As a matter of fact, however, it has been found that vertigo can occur with disease of the external and the middle, as well as the internal ear. An undoubted case of idiopathic or primary labyrinthine disease has not been observed, and most of the cases which have occurred have been found to be due to extension inward from the brain or from the middle ear. Thus, meningitis either of the epidemic or sporadic cerebro-spinal type, purulent meningitis, and fracture of the base, particularly that involving the petrous portion of the temporal bone, have set up labyrinthine trouble. From the middle ear the labyrinth can be affected either by direct extension of disease into it, by pressure inward upon the labyrinth through the apertures between the middle and the internal ear, by products of inflammation within the middle ear, or by a closure of the Eustachian tube which admits air into the ear-drum and the membrana tympani, thus causing collapse of this membrane, and thereby slight pressure upon the labyrinth. Even disease of the external ear, if it be sufficient to exert pressure inward through the middle ear upon the fenestra ovalis and rotunda, may cause the peculiar symptoms. Ménière's disease, therefore, as it is now understood, is really a complex of symptoms due to disease of the external, middle, or internal ear. The salient symptom is vertigo, and this varies somewhat in degree and kind according to which portions of the ear are affected. In labyrinthine disease the patient staggers to one side, the vertigo is apt to come on in paroxysms, and these vary in intensity, so that the patient may simply reel like a drunken man, or may fall suddenly as if shot. Incredible as the history may seem, I once had a patient under my care with this sensational record: He had been a peasant in Ireland, and one day crossing one of the wide moors in a dogcart, he was suddenly, as he thought, struck a violent blow from behind, so that he believes he lost consciousness for some time. At all events, when he was able to get up, he found his horse and wagon some distance off, and, of course, not a soul in sight. Under the belief that he had been struck by some enemy, he went quietly home and said nothing about it. Some time afterward, however, in crossing another lonely place he had a similar experience, and as he came to the conclusion that nobody could have been near him, he made up his mind that it was some malevolent stroke of the

devil, and he consulted a priest, who agreed with him in his belief and gave him an amulet to wear. A series of similar attacks occurred, and, puzzled as to whether there was some diabolical agency at work, or whether he was the victim of a conspiracy, he emigrated to America, and had no attacks for several months. When a new paroxysm occurred he came to me, and I found indubitable evidence of labyrinthine disease. These paroxysms are usually accompanied by nausea or vomiting. The face grows pale and the body is often covered with a cold sweat, so that the attacks are frequently mistaken for apoplexy. In disease of the middle ear the vertigo does not occur in paroxysms, but is apt to be continuous. In disease both of the middle and internal ear loud noises are generally heard, and sometimes these sound like bells or pistol-shots, or loud hissing sounds like steam-whistles. In disease of the external ear the vertigo is less in degree and continuous, and the loud sounds are not usually heard.

The pathology of Ménière's disease, as would be expected from the foregoing remarks, is a complex one, involving lesions of the internal, middle, and external ear, and possibly sometimes, of the temporal lobe of the brain. In labyrinthine disease there may have been found an extension inward of a meningitis or a necrosis of bone. In middle-ear disease there will often be found to be old lesions dating back to scarlatina, measles, variola, and other childish diseases, or there may be a purulent inflammation, or a closure of the fenestra ovalis or rotunda, or the Eustachian tube may be found to be impervious. In disease of the external ear there may be a simple otitis externa or an accumulation of wax or a furuncle. Flourens, in 1828, demonstrated that section of the semicircular canals of the labyrinth in pigeons caused peculiar pendulum-like movements of the head, which ceased a short time after one-sided section, but which in bilateral section were always in the direction of the injured canal, so that after section of both horizontal canals the pendulum-like movement of the head was in a horizontal direction from one side to the other, and after section of both vertical canals in a vertical direction from above downward. Answering to the movements of the head, the pigeons, after section of the horizontal canals, moved to the right or to the left in a circle, whilst after section of the vertical canals they frequently stumbled forward or backward. The ability to fly was seriously disturbed in all instances after bilateral section, and even on a level surface the birds moved with difficulty. The hearing of birds so operated upon was preserved. These experiments were confirmed by Harless, Czermak, Brown-Séquard, and Vulpian. Goltz added some further important facts. He observed in pigeons, in which the semicircular canals were destroyed, a rotation of the head to 180°, so that the hinder part of the head was twisted around to the floor and the beak pointed upward; at the same time there were pendulum-like movements of the body, often movements backward or retropulsion, and loss of ability to fly. Goltz believed that disturbances of movement of the body were due to the defective position of the head alone, for when the

head was fixed in an abnormal position in pigeons that had not been operated upon similar disturbances of the body were seen. He also affirmed that this abnormal position of the head was due to the loss of the semicircular canals, and he therefore regarded these latter structures as sensory organs necessary to the equilibrium of the body, especially of the head. Böttcher advanced the idea that the disturbances of equilibrium were due to a cerebral lesion, complicating and following the operation upon the semicircular canals, and that this lesion was induced by the operative procedure injuring the cerebrum or the cerebellar peduncles. Baginsky claims to have confirmed this view, although he details no experiments. A number of very interesting researches were published a few years ago by Ewald upon pigeons, daws, cockatoos, salamanders, frogs, rabbits, and dogs; as a result of which he arrives at the conclusion that the labyrinth consists of two functionally distinct apparatuses, namely: the auditory labyrinth and the tonus labyrinth. The auditory labyrinth is the true auditory organ, and is that which is irritated by waves of sound, and its excitation passes to the sound-receiving portions through the trunk of the auditory nerve. The tonus labyrinth is the organ which exercises influence upon the muscular movements, so that its excitation is made evident by muscular disturbances which are not as yet thoroughly understood. Heretofore it has been assumed that the auditory labyrinth necessarily converted sound into nerve-irritation, but Ewald's experiments seem to show that this auditory labyrinth simply serves the purpose of directing the sound according to its quality to different fibres of the auditory nerve. Ewald endeavors to make this plain by comparing the labyrinth to the retina, which only responds to light in certain locations; with this difference, however, that whilst the optic fibres are not directly responsive to light impinging upon them, the auditory fibres respond directly to sound. In the higher vertebrates the auditory labyrinth is found in that portion which is known as the cochlea, so that widespread disturbance of the other portions of the labyrinth would not prevent the animal from hearing perfectly, although it might present symptoms of muscular disturbance; and conversely, a lesion of the cochlea might impair or destroy the hearing-power, yet there be no muscular disturbance present. In one pigeon, for example, the whole labyrinth on the left was removed, and on the right only the cochlea, in both operations the naked stump of the cochlear nerve being seen in the amputated cochlear excavation. When this pigeon was suspended the right wing hung motionless, whilst the left was drawn up to the body. If a noise was made, as by clapping the hands or tapping with a hammer upon an adjacent table, the right wing was drawn up more and more until it hugged the body as closely as the left one. As the pigeon did not possess a cochlea upon either side, and as it remained as irresponsive to sounds as a pigeon without either labyrinth, the conclusion is a fair one that the movement of the wing that has been described was not in conscious response to the sound, but rather in response to the excitation set up in the tonus labyrinth by the sound. As Ewald states, this would explain the

well-known fact that certain rhythmical sounds have a tendency to excite to rhythmical muscular movements, exemplified in the desire to dance that is aroused by the hearing of dancing music, or the freshening of the step of tired troops by marching airs. Indeed, Ewald shows that there need not be a rhythm in the sound in order to excite muscular movements. He cites a curious observation of his own, which was accidentally made during some observations in the registration of the normal respiratory curve. In a room in which sat persons to be observed, breathing quietly, there happened to hang a pendulum that ticked off every second with a short simple stroke, and quite unconsciously most of the individuals in this room began to breathe after a little while in exact accordance with the rhythm of the pendulum, twenty times in every minute. Similar are the movements with which an equestrian accommodates himself to the gait of his horse. When both labyrinths are removed from the animal, certain peculiar disturbances become evident in the use of the striated muscles, consisting chiefly of the lack of precision, which may be due to the fact that the contraction of the muscles begins too late, or proceeds too tardily, or is due to lack of strength, etc. Which of these factors it is that causes the lack of precision Ewald has not been able to determine, although he has often found the muscular strength diminished. Those muscles which require the most precision in the performance of their normal function suffer the greatest from the removal of the labyrinth, and therefore the ocular muscles are apt to be most affected, next the muscles of the neck and head, which are especially used in many birds in directing the beak to pick up the food; whilst the wings in birds suffer less and the limbs still less, although in the cockatoo, which uses its leg- and foot-muscles in grasping its prey, the muscles of the lower extremities become very greatly disturbed in their precision of movement. Although Ewald is of the opinion that the labyrinth innervates all the muscles of the body, he has found that in pigeons lesion of it is more apt to impair certain muscular groups, so that he believes that in these birds it is the muscles moving the vertebral column and the head which are most affected, always of the opposite side; yet, curiously enough, he finds that it is different with regard to the extensors and abductors of the extremities, on the one hand, and the flexors and adductors on the other, as the labyrinth is in closer relation with the former upon the same side, and with the latter upon the opposite. The ocular muscles, too, seemingly with the exception of the rectus externus, are mainly innervated from the labyrinth upon the same side. Removal of the labyrinth in these birds causes, therefore, a spiral deviation of the entire vertebral column toward the side operated upon, and of the head and the neck to the same side, whilst the extremities of the same side are flexed and adducted, and those of the opposite side extended and abducted. The muscles of the eyes, especially those upon the same side, deviate toward the side operated upon. Ewald regards these muscular disturbances, however, as of indirect origin, because they vary qualitatively and quantitatively from moment to

moment until death occurs, and also because excitation of the auditory nerve causes them to disappear. He therefore believes that from the labyrinth flows out a continuous innervation to the muscles, a sort of nerve-tonus, and hence he calls this portion of the labyrinth the tonus-labyrinth, locating it in the crista ampullarum and the macula acousticæ.

Diagnosis. The diagnosis of Ménière's disease must be made with the object of first distinguishing it from other forms of vertigo, determining whether it is the external, middle, or internal ear that is affected, or whether there is a lesion of the temporal lobe of the cerebrum or of the cerebellum and its peduncles. The differentiation of Ménière's disease from other forms of vertigo has been gone over in considerable detail in the section upon "Vertigo," and it is not necessary to repeat here what has there been said.

Disease of the external ear can be readily ascertained by the ordinary examination, as well as by the fact that the vertigo is apt to be slight in degree and not continuous, and loud sounds are not generally heard. When there is disease of the middle ear the vertigo does not occur in paroxysms, but is apt to be continuous, and the diagnosis can be made by exclusion of internal and external ear disease, lesion of the cerebrum, or of the cerebellum and its peduncles. Lesions of the middle ear are especially apt to occur in conjunction with a naso-pharyngeal catarrh extending up the Eustachian tube or occluding it, and in all these cases a careful examination should be made of the rhino-pharynx. In disease of both the middle and internal ear loud noises are generally heard, frequently sounding like bells or pistol-shots, or loud whistling sounds resembling steam-whistles. Again, in disease of the middle and internal ear there is a tendency to much more marked disturbance of equilibrium than in case of external-ear disease, and these are such that the patient staggers to one side or the other, or has a sensation as if he had been struck a severe blow, as in the case I have narrated, or is at times moved in a semicircle, as in disease of the cerebellar peduncles. It should, however, always be borne in mind that disease of the middle ear by pressure upon the fenestra ovalis or rotunda may cause the same symptoms as disease of the internal ear does. Disease of the mastoid cells, especially abscess, may also cause the symptoms of lesion of the internal ear. Reference is also made upon p. 160 to other means of diagnosis of disease of the external, middle, and internal ear. Moreover, according to Ewald, as we have seen, a muscular inco-ordination may be present in disease of the internal ear, quite distinct from the disturbance of hearing.

Prognosis. The prognosis of Ménière's disease will depend upon whether it is due to lesion of the external, the middle, or the internal ear. Labyrinthine disease is a grave matter, and although there are cases which can be relieved, there are very few of them. Disease of the middle ear due to chronic lesions of long date are incurable, but some cases of recent lesions can be treated very successfully. Disease of the external ear can usually be treated successfully, and is therefore of good prognosis.

TTEATMENT. The treatment of Ménière's disease will vary according to the cause. Disease of the external ear can usually be treated with success by the ordinary means. Disease of the middle ear can be treated, if I may judge by the experience which I have had in a number of cases, with much greater success than aurists generally suppose. I am firmly convinced that much more can be done in many of these cases by treatment through the rhino-pharynx than by treatment through the external ear. I have again and again seen cases of middle-ear disease to which no relief had been given by the aurists through the ordinary methods, and which have been entirely or largely relieved by removal of a naso-pharyngeal catarrh, and in several of these cases the catarrh has been so slight that the laryngologist himself was in doubt as to whether it could possibly affect the middle ear. Dilatation of the Eustachian tube by the ordinary catheter is, I am quite persuaded, not only useless in such cases, but it is positively harmful, inasmuch as it will increase the vertigo and other nervous symptoms. The best treatment will be by means of suitable sprays projected by the modern compressed-air apparatus of the laryngologist, and not by the ineffective hand-bulbs which are in frequent use, never by means of the antiquated insufflation of powders, or salt and water, which are, I am sorry to say, too often recommended. I am fully persuaded that the aurists have neglected the naso-pharyngeal treatment of middle-ear disease inducing the Ménière symptoms, and I am never satisfied with the prognosis of the aurist in a case of this kind unless I know that he is moderately skilful in the treatment of naso-pharyngeal disease. In cases of disease of the middle ear it is often very difficult to do much, but that a great deal more can be done than has been generally supposed has been demonstrated by the two remarkable cases which Burnett has reported, in which he removed the membrana tympani and the malleus, and completely relieved the nervous symptoms. Charcot has recommended the use of large doses of sulphate of quinine in Ménière's disease, but this was done at a period when we were scarcely acquainted at all with the different forms of this complex symptom-group, and it is certain that it more frequently does harm than good; indeed, I consider quinine in large doses in any of these cases a very dangerous remedy, and I have in two instances seen permanent deafness produced by it. The bromide of potash in moderate doses will be serviceable in combating the nervous erythism which often accompanies the local aural symptoms, but it should be used with great caution, and the patient should not be allowed to form the bromide-habit. Tonics should be given whenever they are needed, but they should be in the form of ordinary bark, iron, arsenic, or the simple bitter tonics, and never in the form of quinine. When there is a purulent disease of the mastoid trephining should be done promptly, as the operation is a perfectly harmless one in this day of antiseptic surgery, whilst the results of purulent extension to the middle ear cannot be foreseen except by omniscience. A lesion of the temporal lobe should be operated upon if it can be localized, and the same is true of a lesion of the cerebellum. Indeed, in any

case where there is grave doubt as to whether a suppurative meningitis or a cerebral abscess has been set up, trephining should be done, because such an operation is not a dangerous one in these days of antiseptic surgery, being of much less risk to life than pus pent up in the cranial cavity. But instead of mere peep-holes being bored in the skull, as it is the custom to do, a large flap of bone should be removed, as advised in the section on "Intracranial Growths," so that a goodly surface may be exposed. Moreover, careful but extensive probing with a small ball-headed probe should be done, if the abscess is not readily found. It must, however, be borne in mind that purulent extension inward from the ear may cause venous thrombosis and lymphangitis, so that in the former case it would be impossible to afford relief by any operative procedure, whilst in the latter an abscess might form in some region far away from the ear—indeed, often in the posterior fossa of the skull, or in the cerebellum, or there might be multiple abscesses. (See Chapter, "Thrombosis of Intracranial Sinuses.") Trephining, therefore, should always be regarded as an exploratory measure, and this should be made known to the patient's friends or relatives.

CHAPTER XVI.

FRIEDREICH'S DISEASE.

Synonyms: Hereditary ataxia. Hereditary ataxic paraplegia (Gowers).

This malady was first described by Friedreich in 1861.

The symptoms begin in early life, and consist of ataxia, nystagmus, defective speech, loss of knee-jerk, and curvature of the spinal column. Ataxia of the lower limbs is almost invariably the first symptom; then of the trunk and the arms. The nystagmus is bilateral, usually transverse, and is generally observed only when the patient fixes or follows an object; but it should be borne in mind that it is not one of the early symptoms. The defect of speech is due to a defect of articulation which causes the patient to stammer, run the letters together, or make sounds that are not precise and that are evidently due to ataxia of the muscles of articulation; and speech may finally become utterly unintelligible. The knee-jerk is always absent. The spinal curvature is usually a combination of lateral and angular curvature, but either one of these varieties may be present alone. It is probably due to ataxic implication of the vertebral muscles. In addition to the cardinal symptoms that have been enumerated there is one mentioned by Rutimeyer that may prove to be of some importance, namely, a turning up of the great toes toward the dorsum of the foot, although Ormerod has seen the same symptom in a case of long-standing multiple neuritis. The gait is a stamping, stumbling, tottering one, and when standing with the eyes closed and feet approximated (so-called Romberg symptom) the patient sways. There is often inco-ordination in the upper extremities, as has already been said, although it is most marked in the lower extremities. There is frequently a jerky shaking of the head and quivering of the trunk-muscles, even when the body is at rest, and jerky tremors of the fingers or of the whole body. Sensory symptoms are usually absent, although in some cases, after the disease has been well developed, severe pains have been observed and diminution of sensibility. There is never any mental disturbance. The progress of the malady is exceedingly slow. Some cases have lasted thirty-three years, although others have terminated fatally in eight.

The disease is an eminently hereditary one in the sense that the families in which it occurs are almost invariably prone to neuroses. It has been a question whether the ataxia itself is ever directly hereditary, but Vizioli has been able to decide this most positively in the affirmative, for he tells of a family in which an ancestor begat eight ataxic children, one of which cases this author diagnosed as

Friedreich's disease, and this patient in his turn begat two children, both of whom were, under Vizioli's observation, afflicted with Friedreich's disease. Nevertheless it does sometimes occur without heredity, and Ormerod alone records three such cases, and I have myself seen two. A very important characteristic of the hereditary tendency is that many members are generally attacked in one generation, and Bury, writing in 1886, goes so far as to say that there are only four instances where one member alone has been affected in a generation. It is essentially a disease of early life, usually beginning between the fifth and fifteenth years, somewhat earlier in boys than in girls, and in some cases so early that the patients have never learned to walk. The exciting causes have been numerous; thus it has occurred during convalescence from scarlet fever and typhus, after chorea, smallpox, migraine, gastric disturbance, and several acute diseases, although it is possible that in a number of these the febrile symptoms have really been due to the disease itself.

In all but one of the cases that have been examined pathologically, some nine in all, the principal lesion has been a sclerosis of the posterior columns of the cord, most marked in the lumbar region and least so in the cervical, in the former affecting both the columns of Goll and of Burdach, in the latter chiefly the columns of Goll. In Friedreich's cases the peripheral portion of these posterior columns was most affected, while in Brousse's the neighborhood of the central canal and the edge of the posterior cornua were most implicated. The pia over the posterior columns is usually thickened and pigmented, and there are fine adhesions between it and the dura mater. In one of Friedreich's cases the posterior columns alone were affected, but this isolation to one system of fibres is unique, and in most the lateral and the anterior columns are affected as well as the posterior. The gray matter of the cord is almost invariably implicated. In one case there were two canals symmetrically placed on each side of the junction of the posterior and anterior cornua; in another there was atrophy of some of the cells of the anterior cornua; in three the cells of Clark's column were degenerated and signs of inflammation were present about the central canal. The posterior roots of the cord are generally atrophied and sclerosed. Slight changes have been observed in the medulla, mainly in the posterior pyramids as high as the nucleus of the hypoglossal. In one case of Friedreich's there was atrophy of the sciatic and hypoglossal nerves, and to a less degree of the anterior crural and median nerves. Until within a very recent date the pathological changes have been thought to be entirely spinal, but a remarkable autopsy has recently been published by Menzel. The patient was undoubtedly a case of hereditary ataxia, and one of his children was afflicted with the same disease. Macroscopically a high degree of atrophy of the cerebellum and pons was observed. In the lumbar cord there was degeneration of the posterior columns of the lateral pyramidal columns and the place of the lost nerve-fibres was taken by connective-tissue septa. The number of the nerve fibres in the posterior roots was considerably diminished. The rim-zone of Lissauer was normal, but in

the spongy portion of the substantia gelatinosa almost all the nerve-fibres had disappeared, only a few being visible, coming directly from the posterior roots, and certain others on the edge of the lateral columns. In the substantia gelatinosa itself the fine nerve-fibres had almost entirely disappeared, and none were visible coming from the posterior column, although a few were still present of the coarser fibres from the posterior roots. Clark's columns were intact. The fibres passing from the posterior columns into the anterior cornua and traversing the spongy substance were normal. The posterior gray commissure showed very few medullated nerve-fibres, although the anterior gray commissure was normal. In the anterior cornua the gray cells were somewhat diminished in number and size, and the anterior roots were partly atrophic. The posterior root-ganglia were macroscopically diminished in size, and microscopically showed great diminution of medullated nerve-fibre. In the lower dorsal cord there was degeneration of the columns of Goll and Burdach, the lateral pyramidal columns, and the direct cerebellar columns. In this region the fibres passing from the root-zone of the posterior columns into the anterior cornua had almost entirely disappeared, and the cells of Clark's columns had undergone fatty and pigmentary degeneration, whilst the fibres from the posterior columns into Clark's columns and from the direct cerebellar columns into them had disappeared, as well as those fibres coming from the anterior cornua and the region of the anterior commissure to Clark's columns. In the anterior horns the ganglion-cells were atrophic, and the anterior roots were also atrophic. The upper dorsal cord displayed the same changes. In the cervical cord there was degeneration of Goll's columns, the columns of Burdach, the lateral pyramidal column, and the direct cerebellar column, although both of these latter were but slightly affected. There was no degeneration of the anterior columns in this region. In other respects the cervical cord was as the lumbar and dorsal. The medulla oblongata was macroscopically much reduced in size. The nuclei of the columns of Goll and of the columns of Burdach, especially the outer nucleus of the latter, were greatly atrophied and diminished in the number of their cells, and were half of the normal size. On one side the territory which Darkschewitz has regarded as the origin of the spinal accessory was much atrophied, and atrophic fibres were found in the trunk of the spinal accessory of this side. The nucleus of the lateral columns was also markedly atrophied, and also the larger olives. The inter-olivary layer and the fibræ acutæ internæ were normal or almost normal. The pyramids were normal. The direct cerebellar columns were so degenerated that scarcely any medullated fibres were to be seen. In the nucleus of the hypoglossal was observed great atrophy of the cells, and the hypoglossal root was markedly degenerated. The restiform body, or inferior cerebellar peduncle, was diminished in size to one-fourth. The facial, auditory, abducens, and sensory trigeminal nuclei were all normal. At the level of the superficial origin of the fourth through the pons the latter was so much diminished in size as to be only two-

thirds of its normal volume, and this diminution of size was due to atrophy of the medullated fibres in the cerebellar peduncles and to entire loss of the large nucleus of the pons. In the middle of the pons the lemniscus was fairly normal, but the lateral lemniscus presented a slight atrophy. The central field of the tegmentum was normal, but the region behind it was so atrophic that the pyramids at this level were surrounded by but few medullated nerve-fibres. The superior cerebellar peduncles were normal. In the anterior and posterior quadrigeminal region there was nothing abnormal. The pes of the crura cerebri was diminished one-third. The fourth ventricle and the aqueduct of Sylvius were found to be considerably enlarged. The cerebellum was markedly atrophied. The fleece surrounding the corpus dentatum was somewhat diminished in the number of its medullated fibres, the nucleus of the corpus dentatum was also somewhat degenerated, and still more marked was the atrophy of the nerve-fibres of the individual lobules, and in these atrophic places connective-tissue septa had taken the place of the nerve-fibres. In the cerebellar cortex there was a diminution in the number of the large cells of Purkinje, and, rather curiously, where the cells had disappeared they had disappeared entirely and no degenerated ones were found. The upper portions of the cerebellum were more atrophied than the lower portion. The medullated nerve-fibres from the direct cerebellar column could be followed in places directly into the medullary portion of the cerebellum. The atrophy of the restiform body and direct cerebellar column in this case of Menzel's is confirmatory of Von Gudden's experiments that Morchi had already corroborated; and a number of cases of atrophy of the cerebellum have been reported (Huppert, Schulze, Frazer, Seppilli, Kirchhoff, Claus, Ferrier); but this case of Menzel's is the first instance of a clinical history having been carefully studied in the cerebellar atrophy of children and the first case in which its relations to Friedreich's disease have been shown. The truth of the matter probably is that Friedreich's disease is closely akin to locomotor ataxia. In all departments of medicine we have the same tissues afflicted by similar diseases with different clinical symptoms, and this is probably the case with locomotor ataxia and Friedreich's disease. Indeed, as Erb says, borrowing botanical language, the natural order of tabes contains two species to which most individuals may be referred; and there are transitional forms, as in Corre's case, in which there was well-marked heredity and affection of speech, yet the disease began with pain and numbness in the feet and legs, and diplopia was observed; and Dreshfeld has reported three cases which would be indistinguishable from locomotor ataxia were it not for the fact that two other members of the same family were affected.

The diagnosis of Friedreich's disease is from locomotor ataxia, disseminated sclerosis, and ataxic paraplegia. In locomotor ataxia the disease usually commences later in life; there is much more apt to be a history of syphilis than a marked heredity; there are frequently early bladder-symptoms, peculiar stabbing and lightning pains, and other impairment of sensation, such as anæsthesia, anal-

gesia, etc., and the ataxia is often in the lower extremities and of a peculiar stamping kind. Moreover, in many cases of locomotor ataxia there are the pupillary symptoms, the transient paralyses of the ocular muscles, and the optic-nerve atrophy. In disseminated sclerosis there is almost invariably a tremor, and this tremor is fine and does not consist of large jerky, shaking movements, which are sometimes seen in Friedreich's disease. It must be admitted, however, that when disseminated sclerosis occurs in children it may be very difficult to make the diagnosis, inasmuch as disseminated sclerosis may be attended by a loss of the knee-jerk, and among its usual symptoms is a nystagmus; but in such cases as these the history of ataxia in the family, or of some disease resembling it, may prove to be of great diagnostic value. From ataxic paraplegia the disease can generally be distinguished, because in the former there is increase of knee-jerk and ankle-clonus, and because the nystagmus and articulation of the latter do not occur. In some cases of ataxic paraplegia, however, when the knee-jerk is lost, the diagnosis may become very difficult.

The prognosis of Friedreich's disease is very serious so far as regards arresting the malady, but, as has already been said, it may last over thirty years before proving fatal.

Nothing is known about the treatment. I have never been able to treat a case myself for any length of time, and therefore cannot speak of it; but if I were to treat one, I should treat it upon the same principle as I would a case of locomotor ataxia.

CHAPTER XVII.

MYXŒDEMA.

History. In 1873 Sir William Gull read a paper upon "A Cretinoid State Supervening in Adult Life in Women," describing very fully the disease now generally recognized as myxœdema, not, however, dealing with the pathology. In 1877 Dr. Ord presented a communication to the Royal Medical and Chirurgical Society of London on "Myxœdema, a Term Proposed to be Applied to an Essential Condition in the Cretinoid Affection Occasionally Found in Middle-aged Women," containing observations that went back as far as 1861, and dealing quite fully with the pathology. Many papers have since been published upon this subject, notably that by Charcot, and by Reverdin, Kocher, Semon, Burns, Horsley, and Virchow. But by all odds the most exhaustive communication upon the subject is the report of a committee of the Clinical Society of London that was nominated in 1883 and reported in 1888.

Clinical History. In this disease the whole body is swollen, this swelling usually appearing first in the head and face, whilst the lower extremities are apt to be swollen before the upper ones. The features become coarse and puffy, the eyelids are always transparently swollen, the eyebrows generally raised to help in sustaining the upper lid, the nostrils are swollen and broadened, the lower lip thickened, everted and livid, and the mouth widened transversely. Over the cheeks and nose there is a reddish patch in the midst of the pallid skin, which is dry, scaly, and without perspiration or sebaceous secretions. The hands are generally affected, the fingers being swollen and moved with difficulty. The hair is dry and brittle, and baldness generally supervenes, whilst the eyebrows and lashes may also be lost. The feet are affected in the same way as are the hands. The temperature is usually subnormal. Œdema of slight degree is occasionally observed, especially about the ankles, but persistent dropsy is seldom seen. There is no impairment of sensation, except in the way of retardation, which has been observed in about half the cases, possibly due to the hebetude. Dysæsthesia and paræsthesia are not infrequent, such as a feeling of coldness, trickling of water upon the skin, disagreeable smells and taste, partial numbness, noises in the ears, vertigo, tinnitus, and pains and heat at top of the head. In a certain proportion of cases there is considerable occipital headache. Paresis of the muscles of the head, neck, and limbs is frequent, so that the head falls forward, the chin resting on the sternum as in the attitude of the *crétin*, and slowness of movement is characteristic, being rarely absent from the gait. Inco-ordination is also occa-

sionally observed. The mental changes consist of sluggishness, dulness, and abnormal persistence of thought and action, with marked irritability. Memory is also impaired at an early period of the disease. Speech becomes difficult and deliberate. Sleep is usually good, but occasionally there are horrible dreams and odd sensations at night. Hearing is usually impaired. The sense of smell is not infrequently defective, but taste is still more often affected. The heart is usually normal, the pulse soft and slow, whilst the capillaries of the face are nearly always dilated. In 59 cases out of 109 analyzed by the committee of the London Clinical Society, the thyroid gland was not altered in size in 23, but it must be borne in mind that the swelling of the neck makes it very difficult to examine this gland. In 22 of these cases the gland was atrophied or reduced in size, and in 3 of them previous enlargement was mentioned. Respiratory troubles are infrequent. The urine is usually lessened in quantity; albumin is present in the later stages, and the excretion of urine is decreased, whilst frequency of micturition and incontinence have been described. The progress of the disease is usually very slow, and some cases have remained for ten years under observation.

Etiology. Most cases occur between the ages of thirty and sixty-five, but the disease may begin at almost any age. Women are principally affected in the proportion of six females to one male. The disease has also been hereditary in some females. As myxœdema is so pre-eminently a disease of women, it has been supposed that it would bear some relation to the catamenia; but this belief has not been confirmed, and it has even been found that myxœdema does not prevent pregnancy; so that this may even occur and go on to the full period in the advanced stage of the disease. In a few of such cases the children died of phthisis, and in one child there was a suspicion of myxœdema. Prolonged lactation, excessive hemorrhage, and acute rheumatism have been supposed to be exciting causes. It was at first believed that myxœdema was a variety of sporadic cretinism, and as such it was first described by Curling and Fagg, of England, and eleven cases are reported from Lower Brittany by Morvan, but not a single record has come from Derbyshire, in which sporadic cretinism has been observed.

Pathology. The pathological alterations which appeared to the naked eye are abundance of subcutaneous fat, occasional emaciation, very slight anasarca, passive effusions into the serous cavities, occasional interstitial changes in the kidneys, occasional hypertrophy of the left ventricle of the heart (in which cases the kidneys are granular), usually atheroma of the large arteries, without any marked degree of endarteritis, in a few instances tubercular disease of organs, and in still fewer instances cirrhosis of the liver. But in every case the thyroid gland was markedly atrophied, usually bilaterally, but in some few cases more markedly so in one lobe than in the other. Microscopically, a series of alterations have been observed. In the skin the coiled tubes of the sweat-glands have swollen epithelium in which nuclear proliferation has occurred, obstructing the lumen,

while in the later stages the nucleated fibrous growth is found in the peri-tubular tissue. Similar, possibly identical, changes occur in the sebaceous glands. Around the hair-follicles there is a nuclear growth, and around this occasionally a nucleated fibrous tissue. In one case there was a peri-neuritis of the nerve-tubules in the subcutaneous tissue. There is often an interstitial nephritis and occasionally tubercular deposit in the kidneys. Occasionally thick fibrous tracts have been found in the liver, or a thickening around the portal vessels, or an excess of pigment in the hepatic walls, with atrophy of them, although the latter are generally unaffected. Early cirrhosis has also been observed. It is seldom that the heart is affected, but when it is the muscle-nuclei are abundant, and between the fibres are numerous free nuclei, and in one case there was a simple excess of pigment in the fibres. Infrequently, also, slight changes have been observed in the lungs, such as early catarrhal pneumonia, emphysema, thickening of the bronchial septa, or miliary tubercular anthracosis. In only one case was there a cellular accumulation, probably tubercular, observed around the vessels of the brain, the patient dying from general tuberculosis. The cerebellum has always been found to be normal. In one case the spinal cord showed nothing more than some thickening of the septa, especially of the posterior columns, and in another case there was some increase of the pia mater. Fibrosis was found in several cases in the superior cervical ganglia of the sympathetic ganglia, and in three others interstitial changes. The lymphatic glands, spleen, pancreas, uterus, intestines, and aorta have been examined, but the only change that has been found was early tubercular infiltration in the small and large intestines in one case, and some atheroma of the aorta in another. In every case, as has been stated, the thyroid gland has been affected. Nearly the whole gland is converted into delicate fibrous tissue, infiltrated with cells, replacing the gland-vesicles. The arterial adventitia is swollen. In a more advanced stage the gland becomes converted into a delicate fibrous tissue, in which groups of small round cells are scattered, evidently the remains of the vesicles, and the gland-structure is replaced with sparse collections of round cells. It is evident, therefore, that myxœdema is due to a destructive affection of the thyroid gland; that changes occur in the skin, mainly in the sebaceous and sweat-glands; that this latter change is of an irritative character; and that interstitial change does not occur in the organs as a generalized condition, although it is occasionally present in the kidneys, liver, heart, and submaxillary glands. In the case in which a chemical analysis was made of the tissues by Ord there was a large excess of mucin in the skin, and from this, as well as from previous observations of less precision, it had been supposed that one of the constant factors of the disease was such an excess of mucin, but in none of the succeeding cases has any such relative excess been found. As Ord's case presented at the time of death the full swelling of myxœdema, it is believed that the subsequent patients had died at a later stage, when there was a trophic process going on, and that in other cases the subcutaneous tissue had become replaced by fat, whilst in

still others the analysis had been vitiated by the keeping of the specimens for a long time in alcohol. The source of mucin in the body is twofold. It results from the degeneration of the protoplasm of epithelial cells, as in the goblet-cells of mucous membrane and in the cells of the glands, such as the submaxillary. In Horsley's experiments on monkeys from which the thyroid had been removed and myxœdema artificially induced, the cells of the parotid secreted a viscid saliva; these were swollen by mucinogen, and a chemical analysis showed that mucin was greatly increased in amount. Mucin also forms a constituent part of the stroma of connective tissue. It is chemically a muco-albuminous material, one of its constituents being mucin, the other a proteid of the globulin class, which resembles a serum-globulin or periglobulin of the blood in its reactions; but in the chemical analysis of myxœdema the mucin only has been estimated, so that, as the committee of the London Society suggests, future analyses should also estimate the globulin. A number of experiments were made at the instance of the committee by Horsley. He extirpated the thyroid gland in a number of animals. Rats and rabbits showed no effect, but carnivora invariably died, death being preceded by myxœdema. In dogs, 95 per cent. so succumbed within a period varying from a few days to rarely as long as two or three months. The symptoms following the removal of the gland are at first dulness and apathy, next fibrillary twitchings in the muscles, then violent convulsions in the extremities and the trunk, with violent dyspnœa, and often semi-coma or coma. Paresis is always found, especially in the voluntary muscles of the limbs, and various disturbances of nutrition. At first the temperature rises, then falls; but it becomes subnormal before death. In the blood the red corpuscles diminish, whilst the leucocytes increase. In one sheep so experimented on by Horsley no marked change was observed for a year and a half; but in May, as the weather was supposed to be becoming milder, the animal was shorn of its thick fleece, and shortly afterward the weather became very cold, when acute myxœdema supervened, and death occurred within a fortnight, whilst post mortem the usual changes were found. This observation is justly considered to be of considerable importance, as showing the effect of changes of temperature. Horsley observed acute and chronic myxœdema in monkeys after complete extirpation of the thyroid; but the acute condition may be averted by keeping the animals in a temperature averaging 90° F., and the average duration of life was thus extended to 125 days instead of 25 days. In such animals the mucin was found to be greatly increased in the skin, in the tendons, the salivary glands, and in the blood. The committee sent out a number of circulars to different surgeons in order to obtain the results of partial or complete extirpation of the thyroid gland, but the answers do not seem to have been satisfactory. Thus Kocher and Reverdin, the former operating on 32 and the latter on 18, have found myxœdema appearing afterward in a large proportion of the individuals; Billroth and Bardeleben, the former operating on 146 individuals and the latter on 15, have found respectively myxœdema in only two

cases. But, as the report shows, many patients died shortly after the operation, others passed away from observation, or were not under observation for a sufficient length of time, whilst it does not appear that accessory thyroid glands might not have remained. These accessory thyroid glands, by the by, are very frequent, and they are found in the neighborhood of the thyroid body. The committee reached the conclusion that there is strong evidence that myxœdema, sporadic cretinism, endemic cretinism, cachexia strumipriva, and the operative myxœdema of animals, are species of one genus, and that the one pathological fact common to all these conditions is the occurrence of morbid processes involving destruction of the thyroid gland.

Diagnosis. The diagnosis will be gone into in detail under "Acromegaly."

Prognosis. The prognosis of myxœdema is unfavorable, as death results chiefly from pulmonary intercurrent maladies, although the patients may die from the exhaustion of the disease itself.

Treatment. The thyroid extracts have effected improvement that has sometimes been marvellous, often lasting for years; and although it as yet a question whether a relapse does not ultimately occur in all cases, they should always be given a fair trial, as no other treatment is of much avail. Thyroid tablets are made by several manufacturing-houses, and if these are of a reliable make they are most convenient. The thyroid gland can, however, be obtained by any butcher, and an alcoholic extract of it is easily made. It is not at all necessary to keep the extract any longer than is necessary for its making, any more than in the case of any other extract, contrary to what certain interested persons would have us believe. Its use must be continued for months. The dose should be the equivalent of one grain of the thyroid gland to begin with in children and three grains in adults, given twice daily. This should be slowly increased until ten grains are reached with a child and twenty with an adult, and even more may be given in some cases; but muscular pain, cardiac attacks, and emaciation must be carefully avoided. If the thyroid extract does not do well, other means must be essayed. In all cases the patient should live in a warm and equable temperature, and it has been found that the regular removal in the winter to southern climates has caused amelioration in the symptoms. This was especially evident in the case of Horsley's sheep and monkeys. Jaborandi and pilocarpine are exceedingly beneficial in myxœdema, from their action upon the skin. Nitroglycerin has also been a useful drug. Tonics, especially iron, quinine, and the hypophosphites, are also valuable.

CHAPTER XVIII.

ACROMEGALY.

Synonym: Marie's malady.

History. This disease was first described by Marie, and for it he suggested the name that heads this chapter, although he points out that similar cases had been observed as far back as the time of Saucerotte-Noël in 1872. Since that time there have been a number of contributions upon the subject.

Etiology. Very little is known of the causation. Women appear to be somewhat more prone to the disease than men; it has in several instances appeared coincidently in attacks of depression; rheumatism, gout, and syphilis have been observed in several of the cases; and this insufficient knowledge is about all we possess of the etiology.

Clinical History. The onset of acromegaly is usually in the hands, although the head may be the part first affected. The hands become of an unusual size, contrasting strongly with the almost normal shape of the other parts of the upper limbs. This excessive development of the hands is of all the tissues—bones, muscles, connective tissue, and skin. The length of the hand, measured from the wrist to the middle finger, is about normal, but the width and thickness are usually enormous, so that for them Marie has suggested the name of battledore hands, and the English have called them spade-like. The consistence of these hypertrophied hands is hard, and there is no pitting, as in œdema. The inter-phalangeal folds and the thenar and hypothenar eminences are increased in size; but the fingers remain straight, and are as large at their tips as at their base. The nails are small in comparison with the size of the corresponding fingers. Although the hypertrophy usually does not involve the arm or forearm, as has been said, these portions may, in certain cases, be affected. In the course of time the head also increases in size in a way that is pathognomonic. The face is chiefly involved, being enlarged especially in the vertical diameter, and becoming elongated and oval, the forehead is low, the orbits are greatly thickened, the borders of the orbit are also increased in size from dilatation of the frontal sinuses surrounding the orbital cavity. The conjunctiva, fat, and muscles of the eye may be also hypertrophied, and exophthalmus may be present. The lids are long, thick, and brown, and in cases of exophthalmus they may not cover the eyes. The nose is generally greatly enlarged in all its dimensions, the alæ being thickened, and the septum may be double its usual thickness. The lips are especially swollen, the lower one most so, so that the latter may even turn downward. The hypertrophy also affects the

tongue; but this usually preserves its normal shape, although it may be so large as to project from the mouth and interfere with the pronunciation of words. The palatine arch, the soft palate, the tonsils, and the fauces are also increased in size, so that the voice becomes guttural and metallic. The uvula is occasionally elongated and enlarged, exciting cough. The chin is an especially prominent feature, being massive, projecting downward and forward, and all the soft structures of the face are thickened. The upper jaw is not nearly so much affected as the lower, which often becomes prognathous, as the result of which the teeth are separated more or less from one another, and the upper dental arch tends to fit inside the lower. The ears are sometimes increased in size. The cranium itself is somewhat increased in its antero-posterior diameter, chiefly owing to dilatation of the frontal sinuses. Usually there is a curvature of the spine in the cervico-dorsal region, the back being slightly humped, the patient not being able to hold the head up, but bending it forward. A slight lordosis may also be present, usually in the lower part of the dorsal and lumbar regions. The vertebral spines have been found hypertrophied in some cases. Almost all the bones of the thorax are also hypertrophied, so that the thorax projects in front, the antero-posterior diameter being increased to a certain extent, apparently at the expense of its transverse diameter, whilst the lower part of the thorax also projects so as to cause a marked prominence upon deep inspiration. The sternum is most affected by the hypertrophy. The xiphoid cartilage becomes less flexible; the clavicles, the ribs, and the costal cartilages become larger. The upper part of the thorax moves slightly in the movements of respiration, which is mainly done by the lower part of the thorax and the abdomen. Hypertrophic changes may also be found in the neck, the cricoid and arytenoid cartilages, the pelvis, the penis, the scrotum, the testes, and the joints, especially those of the knee, wrist, and ankle. The thyroid body is usually diminished in size, although it has never been seen to disappear totally. The mammary glands may be atrophied and soft, whilst the nipple, on the contrary, is apt to be large, and the abdomen is more or less enlarged. There is usually an area of dulness over the upper sternal region, supposed to be due to persistence of the thymus gland; but there is no real foundation for this explanation of the phenomena. The muscles of the limbs are sometimes atrophied and sometimes hypertrophied. In the vascular system important changes have been induced. The heart is enlarged; the arteries are usually rigid, as in the early stages of atheroma; and varicose dilatations of the veins are often observed. Increase of perspiration is usually a symptom provoked by the least exercise, and the sweat may sometimes be disagreeable in odor. Occasionally there is a marked polyuria, with or without glycosuria, and albumin has been occasionally observed. In several cases peptones have been found in the urine. Anæsthesia is very rare. Little cutaneous tumors, pedunculated, the size of a millet- or hemp-seed, reddish or violet-colored, and numerous, are sometimes observed. There may be an intense and persistent headache, and this is usually one of the first symptoms for which the phy-

sician is consulted. This is usually in the occiput, and may be most severe at night. Pains in the bones and in the joints may also be complained of. Disturbances of menstruation are exceedingly frequent, and are early symptoms, being sometimes amenorrhœa at an early age, or suppression of menstruation. Vision is usually affected in a greater or less degree, varying from amblyopia to amaurosis, and definite changes can usually be detected ophthalmoscopically in the retina, or there may be narrowing of the field of vision, or hemianopsia. Occasionally appetite is markedly increased, as may also be thirst. Deafness is also sometimes present, or impairment of the sense of smell or taste. The mental symptoms are variable, consisting of a tendency to melancholia, with suicide, delirium, coma, somnolency, or sleep disturbed by nightmare.

Duration. The disease lasts from ten to thirty years or even more, and is very slowly progressive. Death usually occurs from syncope, probably induced by cerebral compression, but occasionally some intercurrent malady closes the scene.

Pathology. In many cases of acromegaly there has been found a considerable increase of the pituitary body,[1] which is hypertrophied to the size of a pigeon's or hen's egg, or even that of an apple. This hypertrophied mass exerts pressure upon the surrounding organs, such as the olfactory and optic nerves and the crura cerebri, so that in this way are explained the impairment of smell which occasionally results and the frequent neuro-retinitis. It may also exert pressure backward upon the pons and the nerves arising therefrom. The sella Turcica and the pituitary fossa are deformed and enlarged in all their measurements, and the surrounding sinuses are also involved in these pathological alterations, such as the sphenoidal, frontal, thymoid, mastoid, and maxillary. Microscopically the gland-tissue of the pituitary body has been found to be simply hypertrophied. Andriezen's recent researches upon the pituitary gland have seemed to show that it is of great importance in the human economy. The sympathetic ganglia and nerves have also been found to be hypertrophied, and this hypertrophic process affects the bones as well, such as the vertebræ, the clavicles, the iliac bones, and the long bones of the limbs, although in the latter there is neither lengthening nor enlargement, but only increase in size of the ridges, lines, and projections for the insertion of muscles and ligaments; whilst the short bones of the hand and foot are much increased in size. In what way these hypertrophic growths are caused we do not know. The thymus gland is usually lacking.

Diagnosis. The diagnosis of acromegaly must be from—

Myxœdema;
Chronic rheumatism;
Gigantism;
Partial acromegaly;
Hypertrophic pulmonary osteo-arthropathy;
Vasomotor paralysis of the extremities.

[1] This connects the infundibulum with the cerebrum. (See Fig. 40.) It consists of two lobes, the posterior one being really a part of the cerebrum, and known as the *lobus infundibuli.*

In myxœdema the increase in size is due to a swelling limited to the soft tissues, the skin is of a yellow or waxy color, desquamates, and becomes adherent to the subcutaneous tissue; the face is puffed out like a full moon; and there is no prognathism. In acromegaly, however, the skin retains its mobility almost unimpaired, or even has a greater mobility than usual, whilst there is prognathism, lengthening of the face, and the hypertrophy involves not only the soft structures but the bones. In myxœdema there is no curvature or double hump, and the thyroid body is generally atrophied.

In some cases of chronic rheumatism there may be some marked deformities of the fingers and joints resembling acromegaly; but the pain is much more marked; there are the distinct crackling sounds of chronic arthritis, with some functional disturbances, and the progress of the disease is entirely different. Marie has seen certain individuals in whom there was a combination of rickets and rheumatic disease, which might be confounded with acromegaly, inasmuch as there was an increase in the size of the hands and feet, a thickening and eversion of the lower lip, occasionally swelling of the face, and a certain amount of palpebral œdema; but the fingers are distinctly nodular, the hand is bony and deformed, the soft parts do not present the greatly rounded prominences, there is no cervico-dorsal curvature, and no prognathism, and the evolution of the disease is entirely different.

Gigantism has been often confounded with acromegaly, but a careful measurement will show that there is a marked lack of proportion between the size of the extremities and the length of the limb in acromegaly, whilst in gigantism the extremities and the length of the limb are in proportion. Moreover, cases of acromegaly are rather undersized, seldom over six feet, and usually being less than five feet nine inches in height. Frequently, too, in acromegaly the height and the weight, increasing greatly at first, subsequently diminish to an equally marked degree. In gigantism the relative proportions of the face and limbs are preserved. Acromegaly and gigantism may be combined, however, in the same person.

Hypertrophic pulmonary osteo-arthropathy was so called by Marie because almost all the cases had pulmonary lesions, either empyema, neoplastic formations, or bronchitis. In this disease the hands are very greatly enlarged, often more so even than in cases of acromegaly; but they are also deformed. In the fingers it is the last phalanx that is especially affected, and they are of the shape of the legs of a stool, whilst in acromegaly the natural form of the different segments is well preserved. The nail at the end of the deformed finger is also deformed, being greatly widened, lengthened, and especially curved, so that Marie says the thumb observed sideways has a resemblance to the head of a parrot with its curved beak, whilst in acromegaly the nails appear too small for the large phalanges which they cover. The hand, however, is but very slightly altered, and forms a marked contrast to the fingers; whilst in acromegaly the metacarpo-phalangeal region is enormously increased in size. In the wrist there is very great

enlargement, the lower ends of the two bones of the forearms being enlarged in an abrupt manner, the thickening taking place both in the antero-posterior and lateral diameters, and the lower portion of the forearm being as large as at the middle or below the elbow, whilst the wrists are also deformed. In acromegaly there is nothing of this kind; for if the wrist should be larger than normal, its size is still proportionate to the rest of the upper limb, and there is no projection, abrupt enlargement, or deformity. In the feet there is an analogous condition. The toes, especially the last phalanges, are affected as the fingers are, the tarso-metatarsal region showing relatively as little change as in the metacarpo-phalangeal region; whilst the ankle is, like the wrist, enormously, increased and the lower part of the leg is found as large as the middle portion—bearing comparison, as Marie says, with the foot of an elephant. In this form of arthropathy the hypertrophy is mainly in the bones, and not, as in acromegaly, both in the bones and the soft tissues. The joints are also limited in their active and passive movements, the patients being clumsy with their hands, and their elbows presenting a more or less prominent degree of flexion, so that complete extension is impossible. There is a somewhat analogous condition in the knees, hip-joints, and shoulders.

There is a disease known as partial acromegaly, in which there is a considerable hypertrophy of one-half of the body, or of a lower limb on one side and the upper limb and side of the face on the other. It may involve the hand and foot, the fingers or toes, and sometimes one finger and one toe; but there is always true deformity, and the disease is usually congenital, remaining stationary and being generally unilateral. I have, however, seen one case occurring in middle life, and affecting one lower limb.

In vasomotor paralysis of the extremities there is a marked increase in the size of the hands and fingers, a reddish tint with some livid spots on their dorsal surface, occasional pain in these parts, a certain degree of duskiness extending up the arm to the shoulder, and occasionally there is a slight kyphosis of the upper part of the spine; but the hands are not of the shape of those in acromegaly, the fingers being larger at their bases than at their extremities, and there is no oval face, prognathism, hypertrophy of the lips, enlargement of the tongue, hypertrophy of the bones of the face, or alteration in the speech.

Prognosis. The prognosis of acromegaly is always unfavorable, as treatment has never been able to affect the course of the disease.

Treatment. Treatment can only be symptomatic to a triflingly slight degree. Antipyrin and caffeine seem to have been of some use in relieving the headache. The glycosuria may be remedied by treatment by arsenic, alkalies, and dieting.

CHAPTER XIX.

EXOPHTHALMIC GOITRE.

Synonyms: Basedow's disease. Graves's disease.

This disease was first described by Graves, of Dublin, in 1835, and shortly afterward, in 1840, by Basedow, of Germany.

The three cardinal symptoms are: cardiac palpitation with rapid pulse; enlargement of the thyroid gland; and prominence of the eyes, or exophthalmus. The disease may commence with either one of these three symptoms, most frequently with cardiac palpitation and rapid pulse. The pulse generally rises to about 120, but may even mount to 200 beats in a minute. The pulsations are apt to be most marked in the carotids, whilst the radial pulse is usually less affected, presenting a notable contrast to the other vessels. The enlargement of the thyroid gland generally begins after the arterial alterations have been in existence for some time. This enlargement is not usually very great, and may be temporarily increased by exacerbation of the cardiac palpitation and by emotions. The exophthalmus varies very greatly in degree, being sometimes so slight as to be scarcely perceptible, at others so pronounced that the eye is protruded sufficiently to lay bare the attachments of the ocular muscles, or there may even be absolute luxation of the eye. This exophthalmus is bilateral, but not always to the same extent upon both sides. It may cause trophic alterations of the eye, from the lack of the usual protection of the lids. In many cases a symptom is observed that was first described by Von Graefe: If the patient is made to fix the eye upon an object held before him, and attempts to follow this object as it is moved downward, the upper eyelid will not follow the movement of the bulb of the eye, nor will it do so when the object is moved up again; in other words, there is a lack of association in the movements of the bulb and the lid. With these three cardinal symptoms are occasionally shown some minor ones, such as a sense of heat without actual fever, profuse sweating, and sense of throbbing in the head, pulsating noise in the ears, headache, insomnia, and vertigo. Mental depression and muscular tremor are frequent accessory symptoms. Glycosuria has been often observed, sometimes early and sometimes late in the course of the disease. Enlargement of the lymphatic glands or of the tracheal and bronchial glands is occasionally observed. Mental symptoms are occasionally present, such as melancholia, mania, or general paralysis.

Charcot has also seen some cases accompanied by mild paraplegia, without spastic phenomena or lancinating pains. Wolfenden has shown that the resistance of the body to the electric current is lowered in a remarkable manner, as Charcot has pointed out. Thus, in

some fifty healthy persons, he found that the resistance would register in a moderate and fixed current from 4000 to 5000 ohms; whilst in two cases of this disease the resistance was as low as 200 and 300 ohms, and in eight others it varied from 500 to 700 ohms. The author ascribes this diminished resistance to the dilatation of the skin capillaries, which saturate the skin with fluid. Shaw has examined 46 cases with the same results. A bronzing of the skin is occasionally observed. A limitation of the field of vision has been seen in 20 cases by Caste and Wilbrand. Louise Fiske-Bryson has observed diminished chest-expansion in this malady, in a severe case this being one-half inch, sometimes less; and this has been confirmed by Hammond. The onset is generally gradual, and the disease is a chronic one. Remissions and intermissions occasionally occur, and even recoveries, although the latter are rare. When death supervenes, it is usually the result of the cardiac symptoms.

The pathology of this disease is unknown. It would seem probable that the lesion is in the sympathetic nervous system. White found a hemorrhage in the floor of the fourth ventricle in one case which followed pneumonia. Filehne caused increased pulse-rate, often accompanied by exophthalmus and once by struma, in guinea-pigs by destroying the anterior covering of the restiform body. Durdufi has localized the point of lesion in the tuberculum acusticum. Bienfait has, to a certain extent, confirmed Filehne's experiments. Hammar found no pathological change in the cervical portions of the sympathetic nerve, but the spinal cord was not examined. Roosevelt found the medulla, the sympathetic, and the vagus normal. Yet these different findings do not preclude the idea of the sympathetic being the portion of the nervous system that is at fault, both because our present knowledge will not permit more than very limited localization in any one part of the sympathetic, and it is a tenable supposition that a central lesion could indirectly affect a large tract of sympathetic fibres. Moreover, of late years many diseases that were formerly supposed to be entirely due to lesions of the sympathetic have been shown to be really caused by myelitis of the syringomyelitic type of neuritis, especially of the multiple variety. In the heart, dilatation and hypertrophy are usually found in a slight degree, and even mild endocarditis or valvular lesions are occasionally met with. The arteries are dilated and occasionally aneurismal. In the thyroid gland the vessels are enlarged, and the connective tissue is increased in quantity, with occasional cystic or colloid degeneration. In the orbit there is generally a moderate increase in the amount of fat, although this is occasionally normal; and upon rare occasions there are dilatation of the veins, atheroma of the ophthalmic artery, and a fatty condition of the arterial muscles.

The diagnosis of this disease is usually very easy when the three cardinal symptoms are present; but in the early stage, when only one of the symptoms is to be observed, the real nature of the malady may be overlooked. Generally, however, the excessive pulse is the first symptom, and should arouse suspicion. In ordinary goitre there are no cardiac symptoms or exophthalmus, although large goitre of

endemic origin may be associated with rapid pulsation and exophthalmus; in such a case, however, the exophthalmus is generally unilateral.

The prognosis is generally grave, although relief is frequently obtained, especially when treatment is begun early, and sometimes a cure is effected.

The treatment should consist of rest, belladonna, hemp, iodine, carbazotate of ammonium, hydrotherapy, electricity, and surgical operation. The rest should consist of keeping the patient as quiet as possible, both physically and mentally, and, if necessary, by restriction of the expenditure of energy to the degree that may be needed by keeping the patient in bed, by limiting the amount of exercise taken, or by the abolition of all exercise except such as may be necessary for the pursuit of a business or occupation. In administering belladonna, either the fluid extract or atropine should be employed—the former in doses of 1 or 2 minims three or four times daily; the latter in doses of gr. $\frac{1}{100}$ as often. The dose of carbazotate of ammonium should be 1 grain in pill three times daily for the first week; for the second week 2 grains at the same interval, and for the third week, if it can be borne, 3 grains; but the drug can rarely be taken more than three weeks. Iodine can be administered in the form of tincture of iodine, 30 drops to 2 drachms three times daily, or the iodide of potash, 10 to 30 grains three times daily. Electricity, in the form of galvanization of the cervical sympathetic and of the cardiac region, has seemed to be of considerable use in certain cases. Excision of the thyroid gland has been of use in some cases when the disease has not been too fully developed. Digitalis has not been of much value in this affection, and has often been injurious, except sometimes in the paroxysms of palpitation and dyspnœa.

CHAPTER XX.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM.

In former years and until the last ten or fifteen, the sympathetic nervous system was held accountable for a great many diseases whose pathology was shrouded in mystery; but, as year after year has gone by, one disease after another has been found to be due to lesion of the peripheral or central nervous system, and thus exophthalmic goitre, facial hemiatrophy, muscular atrophy, and pseudomuscular hypertrophy have been shown to belong only partially to the domain of the sympathetic, or have been entirely wrested from it. At the present day, therefore, the main diseases of the sympathetic are considered to be—

Certain vasomotor and trophic symptoms of the head and chest;
Diffuse vasomotor symptoms;
Herpes zoster;
Tachycardia;
Vasomotor joint-neuroses;
Angina pectoris;

Angina pectoris and tachycardia belong to general medicine, and the joint-neurosis to surgery, so that they are not pertinent to a textbook of this kind.

The cervical sympathetic can present symptoms of paralysis or irritation, either from direct or indirect injuries. In paralysis the most constant symptom is contraction of the pupil, and less constanly are observed approximation of the eyelids and retraction of the bulb of the eye. Irritation of the cervical sympathetic, which is rare, causes dilatation of the pupil, expansion of the eyelids, and propulsion of the bulb of the eye. Vasomotor symptoms are seldom seen with lesion of the cervical sympathetic, but the ear and cheek of the affected side are sometimes red and warm in cases of paralysis and white and cooler in cases of irritation. Trophic disturbances are occasionally observed, and consist of a slight flattening of the cheek. These sympathetic symptoms, indicating lesion of the cervical sympathetic, have been observed in cases of tumor of the cervical sympathetic itself, or of its rami communicantes between the brachial plexus, and even from affections of the apices of the lungs (which, by the by, are probably the cause of the flush of the cheek in tuberculous subjects), or from fractures, dislocation, or caries of the lower cervical or upper dorsal vertebræ.

The diffuse vasomotor affections are those known as *digiti mortui*, erythromelalgia, Raynaud's disease, and angio-neurotic œdema. Digiti mortui is an affection consisting of a diffused pallor of the

upper extremities, occasionally of the lower extremities. The fingers become white in extreme cases, like those of a corpse, and the surface-temperature is diminished. The pallor is remittent or intermittent, the remissions or intermissions lasting for days or hours. Erythromelalgia, described by Weir Mitchell, is a curious affection of the soles of the feet in which circular redness and pallor are observed, with some hot, burning pain in the part affected. Raynaud's disease consists of paroxysmal attacks of pain, pallor, coldness, numbness, and anæsthesia of the lower extremities, and a gangrenous condition of the toes. Angio-neurotic œdema is generally intermittent, and affects the skin or the mucous membranes of the larynx and pharynx. When the skin is implicated, the swelling is localized, and is about the size of a plate, the skin being sometimes white, sometimes slightly reddened, and generally in proportion to the œdema are sensations of burning, prickling, and stiffness. The extremities are most frequently affected, especially near the joints, but the eyelids, cheeks, and lips, sometimes even the trunk, are occasionally the site. Vomiting, pain, and constipation are frequently attendant symptoms, and occasionally hemorrhages from the gums and the vesical and bronchial mucous membranes, sometimes even hæmoglobinuria.

As has already been said, the tendency is nowadays to regard these so-called sympathetic nervous affections as really due to multiple neuritis, although it is as yet a question whether they do not begin in the sympathetic nerve-fibres running in the peripheral nerves, and secondarily affect the voluntary nerve-fibres; whilst in some other case actual disease of the spinal cord has been found. Thus, some cases of tingling and numbness in the finger-tips, which were formerly regarded as due to lesion of the sympathetic nervous system, have been shown to be due to multiple neuritis; whilst Morvan's disease, an affection akin to Raynaud's disease, which was at first regarded as a malady of the sympathetic, then as a multiple neuritis, has been shown to be due to syringomyelitis.

The prognosis of the diffuse vasomotor affections is generally grave, so far as regards a cure, and that of Raynaud's disease and erythromelalgia is unmitigatedly bad.

The diagnosis of these affections is generally easy, except that in Raynaud's disease a distinction must be made from syringomyelitis, the details of which are given in the chapter upon this subject. The treatment of Raynaud's disease must be amputation, as speedily as possible, before the strength of the patient is exhausted, and before sepsis sets in. Of the other affections treatment should be by means of electricity, ergot, and tonics. The electricity applied should be galvanism, faradism, and the static current. Galvanism should be applied to the spinal cord and to the peripheral nerves. For the former purpose an electrode (Fig. 67) should be applied in the nape of the neck, another (Fig. 68) to the lower dorsal spine, and a current of three to ten milliampères, in sittings of three to five minutes every day or every other day. To the peripheral nerves an electrode (Fig. 76), placed upon the spine about at the point where the affected

nerves have their origin, and another electrode (Fig. 68 or 69) applied over the affected limb, and a current of three to ten milliampères used. The faradic current should be applied by means of the wire brush, and a sufficiently strong current should be passed over the affected limbs for ten to fifteen minutes every day or every other day. If the static current be applied, the sparks should be made use of, and should be vigorously applied to the affected parts.

CHAPTER XXI.

ALCOHOLISM AND MORPHIOMANIA.

The prolonged effects of alcoholism upon the nervous system may be manifested both by mental symptoms and by paralysis. The chief mental symptoms are: delirium, in the form that is technically known as delirium tremens, occasionally a variety of paranoia, sometimes symptoms almost identical with those of general paresis, a chronic meningitis, acute mania or melancholia, acute hallucinatory insanity, or a recurrent insanity known as *oinomania.* The paralysis that is generally associated with alcoholism is that which is due to a multiple neuritis, differing somewhat in its character from the other forms of neuritis of this type.

Delirium tremens is so familiar a disease in every community that it needs only a brief description. It usually follows alcoholic excess, or a short period of abstinence after such, and the exciting cause may be some slight injury or illness. The onset is usually gradual, extending over a few days, and the prodromata consist of insomnia, anorexia, restlessness, and a semi-hallucinatory condition ; and these gradually pass into loquacity and persistent delusions and hallucinations, often maniacal or violent outbreaks. Hallucinations of hearing are the most frequent, are generally very vivid, and are frequently accompanied by hallucinations of sight. The patient becomes very suspicious, and has sometimes marked delusions, often of marital infidelity, which are, however, much more frequent in the chronic or subacute forms of insanity. Occasionally there is an olfactory hallucination. Not only do these hallucinations excite suspicion, but often dread and terror. There is a characteristic tremor, generally observed only upon voluntary motion of the muscles, but occasionally at all times, and it affects the tongue, face, and extremities. In many cases, also, there is that peculiar picking at the bedclothes or clothing, which is termed *carphologia.* With the tremor there are often observed muscular twitching or shock-like contractions of muscles. The face may be flushed or pale, and this condition of flushing or pallor may alternate. The pulse is usually frequent, soft, and large at first; but it becomes smaller as the disease progresses, and in severe cases it may mount up to 140 to 200. There is usually free perspiration. The temperature is usually 100° to 101°, but it may run up to 103° and 105°, and in fatal cases even higher. The tongue is coated, thirst is present, and the urine is scanty if the perspiration is great. There may also be albuminuria. Slight neuro-retinitis has been observed. These symptoms will increase in gravity in the fatal cases, and may be accompanied by complications due to alcoholic disease of other organs, as of the liver, kidneys, or lungs,

so that there may be cirrhosis and fatty degeneration of the liver, nephritis, and pneumonia. Uræmic coma occasionally occurs also, and sometimes, though rarely, meningeal hemorrhage. The delirium lasts for a variable period extending from two to ten days.

In some cases of alcoholic insanity a variety of paranoia is observed, in which there is a delusion of persecution that will frequently, in the course of time, be combined with a delusion of self-exaltation, although the delusion of persecution is much longer in its duration than in ordinary cases of paranoia; and in these cases there is the same logicality or systematization of the delusions, the same keenness of reasoning power, the same preservation of memory and perception that is seen in the ordinary types of this mental disease. It differs from the ordinary cases of non-alcoholic paranoia chiefly in the fact of the delusions having a tendency to weave themselves around some belief of marital infidelity. In none of these cases have I ever seen any hallucinations of hearing or sight.

In many cases of alcoholism there is a fine tremor of the facial muscles, the tongue, and sometimes of the extremities, a thickness of articulation, and a gradually supervening mental dulness or dementia, which are all very similar to those observed in ordinary cases of general paresis; but ordinarily the former disease is somewhat more rapid in its onset, and the pupillary symptoms are more generally lacking. It is of great importance to make a distinction between this form of alcoholic pseudo-general paresis and the true general paresis, because the former is frequently of good prognosis.

A chronic cerebral meningitis is sometimes observed also in alcoholism, and this is prone to affect the dura mater, causing so-called chronic internal pachymeningitis, although it may also affect the pia. The symptoms are those of ordinary meningitis in a chronic form.

Acute alcoholic mania or melancholia does not differ much from the ordinary forms of these mental maladies, and the so-called recurrent insanity known as *oinomania* is characterized by mental and moral deficiency, various excesses, and indecent, absurd, and silly demeanor.

Acute hallucinatory insanity is more frequently observed than any other form of mental trouble except delirium tremens, and has the same characteristics as ordinary hallucinatory insanity, with the exception that there are apt to be vivid hallucinations of hearing, persistent and logical or systematized, and the patient is more apt to pass into a chronic condition, in which the confusion of the hallucinatory insanity passes away or merges into a state resembling chronic paranoia.

The multiple neuritis of alcoholism has been already spoken of. (*Vide* section upon "Multiple Neuritis.")

The prognosis of delirium tremens will depend upon the age of the patient, the amount of previous alcoholic excess, the way in which the patient has borne it, and the complications due to disease of other organs. As with all the narcotics, the effect of alcohol varies greatly in different individuals, so that what may be excess to one individual or a member of one race may be a matter of slight

account to another individual or a member of another race. Usually, the prognosis of the non-complicated cases is good, with proper treatment. The prognosis of alcoholic paranoia is grave, although a certain proportion of cases recover in a period of two or three years. The prognosis of the pseudo-general paresis of alcoholism is also grave; but many cases recover with proper treatment, and therefore the prognosis is by no means the hopeless one that attaches to ordinary general paresis. The progress of alcoholic mania and melancholia is that of ordinary mania and melancholia. The prognosis of the recurrent form of alcoholic insanity known as oinomania is grave, although a certain proportion of cases recover with proper treatment. The prognosis of the hallucinatory insanity of alcoholism is grave, because there is a great tendency to pass into a chronic condition of paranoia, in which the hallucinations of hearing persist for many years; but some few cases do recover. The prognosis of alcoholic multiple neuritis has been already spoken of.

PATHOLOGY. In chronic alcoholism the cortical vessels are enlarged, tortuous, atheromatous, and fatty. The nuclei of the adventitia are numerous, or proliferate, or are fattily degenerated, whilst there is an abundance of Deiters's or scavenger cells to be found in the upper region of the cortex, just beneath the pia. Around the proliferated Deiters's cells is a dense connective tissue, formed by the meshing of the processes of the Deiters's cells. All these changes are most marked immediately beneath the pia. In the epi-cerebral space there are often large quantities of amyloid bodies, which are found in fresh sections from frozen brain, so that they cannot be due to the action of reagents. The fifth layer of motor cells undergo fatty change, extensive degeneration, or absorption, according to Bevan Lewis. The explanation of the selection by alcohol of this site, and of the outermost rim of the cortex simultaneously with the fifth layer, is supposed by Lewis to be that this outermost layer really represents the distribution of the apex processes of the cells in the fifth layer. The long, straight vessels of the cortex are often enormously distended, even showing minute aneurisms. In the lowermost layer of the cortex, the spindle-cell formation, great numbers of the Dieters's or scavenger cells are met with. In the spinal cord of chronic alcoholism there is an increased vascularity, the vessels of the posterior and lateral columns being most affected, and those of the anterior columns the least. These vessels increase greatly in thickness, from hypertrophy of the muscular coat, and the lumen becomes greatly diminished—so that a vessel which measures eighteen millimetres across, and which would normally have a lumen of thirteen millimetres, may have its lumen diminished to five; or one of twenty-seven millimetres across, instead of an approximately normal lumen of twenty-three may have a lumen of only nine. This change in the muscular coat seems to be identical with that of chronic Bright's disease, and is in the nature of a compensatory hypertrophy. Besides these changes of brain and cord in chronic alcoholism, there may be a simple fatty degeneration of the tissues and their nutritive vessels, and this may be associated with interstitial sclerosis.

Diagnosis. The diagnosis of most forms of alcoholic mental disease is generally easy. The history of the case is the most important factor, and there is usually no difficulty in obtaining this. The delirium tremens is readily recognized by the history of the case, conjoined with the symptoms that have been detailed, and this is also true of the other forms that have been mentioned. In the pseudo-general paresis of the alcoholic it is sometimes impossible to make the diagnosis at the outset; but the alcoholic form may be suspected when the onset has been relatively acute, when the pupillary changes are absent or slight, and when there has been a previous attack of the same kind. Of some value in this diagnosis is also the fact that the case of alcoholic pseudo-general paresis does not usually have the characteristic delusions of grandeur; but this must be considered with considerable caution, since many cases of general paresis, as I have already said, are lacking in this symptom. A week or two of treatment, however, will make the diagnosis clear.

Treatment. The treatment of alcoholism should consist, in the first place, of treatment of the disease from which the patient is suffering, and, in the second place, of treatment of the habit itself. The treatment of most forms of alcoholic insanity will not differ from that of the non-alcoholic types of the same kind, with the exception of the cases of delirium tremens, oinomania, and pseudo-general paresis. Delirium tremens should be treated by the bromides, hyoscine, opium, stimulants, and isolation. The bromide should be given in doses varying from 10 grains to 30 grains every three or four hours, with $\frac{1}{200}$ to $\frac{1}{100}$ grain of hyoscine, either the hydrobromate or the hydrochlorate (it makes no difference which), and sulphate of morphine or bimeconate of morphine should be administered hypodermically in doses of $\frac{1}{8}$ grain once or twice a day, or even oftener in severe cases. Opium or morphine by the mouth is usually ineffective. The question of alcoholic stimulation is a very important one in these cases. Usually it may be regarded as unsafe to make any rapid reduction of the whole amount to which the patient has been accustomed whilst the patient is suffering from any mental disease, although generally it will be perfectly proper to reduce this quantity by one-half. By this I do not mean to say that the quantity which the patient has been taking in some excess immediately preceding the outbreak should be taken as the standard, but the quantity to which he has been habitually accustomed for months or years past. The sulphate of quinine, in doses of 3 to 15 grains in the twenty-four hours, will be found an exceedingly useful adjuvant, and it should be given in the form of the tablet triturate freshly prepared or in capsules, and seldom or never in solution with a mineral acid, as this is prone to upset the digestion when long continued. The question of nourishment in these cases often becomes a very serious one, and in many instances it will be impossible to make use of other than such easily digested food as milk, beef-tea, eggs, and koumiss, whilst as the symptoms grow less severe solid food may be gradually added. An excellent method of giving a raw egg in this condition is to get the patient to swallow it whole after the shell has been removed, and it is surprising to see

with what facility patients can be taught to do this. The most rigid isolation of the patient should be practised, so as to insure the most perfect quietude, and the room should be darkened and all noises carefully excluded. As the symptoms disappear and the patient convalesces, the morphine may be reduced first, and often with comparative rapidity, but the bromides and hyoscine should be continued longer and reduced more gradually, whilst the nourishment should be pushed and the stimulation with quinine continued. During this period of convalescence, too, the alcoholic stimulant should be reduced, and in its place should be given an alcoholic malt-extract, frequently repeated, so that the patient may get it three or four hours apart, with some slight nourishing food.

The cases of oinomania, the recurrent insanity that has been described, should be treated in the same way as a case of delirium tremens, with the exception that it will not be necessary to give such quantities of the bromides, opium, or hyoscine.

The cases of alcoholic pseudo-general paresis must be treated differently from the two forms that we have been describing. In the first place, the patient should be put upon a mineral acid, the best of which is the nitromuriatic acid, of which twenty drops in a wineglass of water, three times a day, should be given after meals. Then with this an aloetic should be combined at bedtime, and I have for years been in the habit of using the time-worn prescription of—

℞.—Ext. aloës, Ext. hyoscyami	āā ℈j.
Ext. nucis vomicæ	gr. v.

M. ft. in pil. no. xv. S.—One every day, two hours after the evening meal.

Or a dose of a mineral water, such as Hunyadi or Rubinat, may be given every morning before breakfast. The patient's diet should also consist largely of fish, soups, eggs, fresh fruits, fresh vegetables, farinacea, and custards; and all meat, either of chicken or the red meats, should be absolutely excluded for an initial period of four or five days. At the end of this time chicken or turkey may be allowed once or twice a day, or if the patient craves red meat very much, he may be allowed to eat this once a day. After having treated the patient for four or five days, 10 or 15 grains of the iodide of potash should be given an hour after meals, well diluted in a full tumbler of water or Vichy or Giesshübler. If the patient is seen to be improving markedly at the end of a week, tonic doses of quinine should be administered, two hours after meals, three times a day (the sulphate being used), or a half-ounce of elixir of calisaya. From the beginning of treatment cerebral galvanization will be found to be exceedingly useful, and this should be administered every second day, with a current of two or three milliampères, in sittings of three to five minutes.

The treatment of alcoholic multiple neuritis is that of an ordinary multiple neuritis *plus* the necessity of breaking up the alcoholic habit.

The question of the alcoholic habit is, as we all know, a very

vexed one. On the one hand, it has been considered as a vice, and, on the other, it has been regarded as a disease. The truth lies, as it generally does, between the extremes. A human being who indulges in alcohol to excess, knowing that it is certain to ruin his health, his prospects in life, and the happiness of those most dear to him, is certainly responsible, and alcoholism in his case is, therefore, a vice; but if he persists in becoming a chronic inebriate, and induces structural changes or forms incurable habits that may be transmitted to his children, the strain in these children is a disease as much as is any other hereditary trait, although even in them it differs from most diseases in the fact that it is within the control of the will to a large extent. There is not one of us who could not drink, use opium or cocaine, commit burglary, forge, steal, or do other vicious acts, if it were not that we were restrained by that force within us which theologists call conscience, which metaphysicians call will-power, and which physiologists call inhibition; and although this conscience, inhibition, or will-power, whichever we may choose to term it, may be weakened by hereditary faults, it is seldom entirely lost, and can be exerted in the majority of cases if the individual will make the attempt. Of late years the question of alcoholism has been fashionably and charitably regarded as a disease in all cases; but although I do full honor to the kindliness of the sentiment that is at the bottom of this mode of thinking, I do yet firmly believe that its effect has been to create much maudlin sentimentality upon the subject. Leaving, however, these primary considerations which are necessarily intermingled with the more purely medical ones, I would say that I regard the cure of most cases of chronic alcoholism as likely to be ineffective unless the patient is put under such care as to make it impossible for him to drink, either in a sanitarium or an inebriate asylum. In the last few years a great ado has been made about the alleged cures of alcoholism by the secret methods of certain quacks. The statistics of the results attained by these methods have never been published in such a way as to be verified or as would be done if the possessors of the secret method were strong enough to stand such a crucial test. Nor have the ill results that many practitioners could cite ever been published, nor are they ever likely to be published, because the seal of professional confidence closes the lips of those most competent to speak, whilst the patient himself and his family are silent for obvious reasons. The spread of the metallic tractors of Perkins throughout America and Europe; the grant of a large sum of money by the English Parliament to Joanna Stevens for her wonderful remedy for the cure of stone, that turned out to be a simple alkali of oyster-shells and utterly worthless; the wonderful stories told of the Grotto of Lourdes and the various sacred relics that from time to time attract swarms of devotees; the startling results that were at first claimed but a few years since for Koch's tuberculin, in spite even of that honest scientist, whose words were given a meaning that he had never put into them; the fortunes made by quacks in every community, serving to bribe legislatures, as well as scores and scores of journals that publish

dishonest and lying advertisements on their back pages, at the same time that they prate in their editorial columns about the benefits of civilization—all these have made a history that should teach mankind to place no faith in secret remedies that dare not come into the light of day. It has been alleged that one of these secret remedies, known as the Keeley cure, consists of strychnine, belladonna or atropine, ipecac, caffeine, cocaine, and codeine; but I have no means of knowing whether these statements are correct. It is true that as far back as 1880 Dr. Dobvonravoff claimed to have met with considerable success in curing drunkenness in the Russian army by the injection of the nitrate of strychnine, of which he used gr. $\frac{1}{25}$, once or twice a day. He alleged that in this way he could induce a distaste for alcohol in two or three weeks. In certain cases this would appear to be a fact; but the remedy has never met with much success in the hands of competent men. The bichloride of gold has certainly been entirely unsuccessful with most physicians. If a case of alcoholic habit is to be treated, the alcohol should be gradually reduced, and this can be done at first with considerable ease, and it is generally only when the dose is becoming very small that the patient begins to manifest great nervousness, jactitation, sleeplessness, and sometimes symptoms of one of the alcoholic disorders that have been mentioned. At this period bromide, quinine, strychnine, and hyoscine should be given in the way that has been mentioned in describing the treatment of delirium tremens, grading the dose according to the symptoms. In most of the cases of alcoholism the patient is usually also addicted to the use of cocaine and morphine, and these drugs must also be diminished gradually in the same manner. I have no faith whatever in the sudden reduction of alcohol, cocaine, or morphine, much as it has been vaunted of late years, and my lack of faith comes from the fact that I have seen a number of cases in which the sudden reduction has seemed to meet with charming success for a short period, to be followed by very serious symptoms, such as some form of alcoholic insanity, or even a convulsion.

CHAPTER XXII.

RAILWAY INJURIES AND OTHER TRAUMATIC DISEASES OF MEDICO-LEGAL INTEREST.

Traumatic diseases are of such vast pecuniary importance in this modern age of great railway and steamship companies and other wealthy corporations that they have been studied with more assiduity than scientific exactitude. Although the older works upon surgery discussed the question of cerebral and spinal concussion, they did so much more from a traditional standpoint than as a result of the personal observation of the authors. Erichsen's first lectures in 1866, on *Certain Obscure Injuries of the Nervous System commonly met with as the Results of Shocks of the Body received in Collisions on Railways*, marked a new era in the treatment of this subject. This was followed by his treatise on *Concussion of the Spine*, in 1875. Page's book on *Injuries of the Spine and Spinal Cord*, published in 1883, was in the same vein; and these three publications, together with the rapidly growing interest of the subject, have given rise to a great literature, of which the most salient articles have been those of Knapp, Putnam, Walton, Oppenheim, Charcot, Clevenger, and Judge Bartlett. During the twenty-five years that have elapsed since Erichsen first made known his views, the study of the phenomena following traumatism has gradually passed from the surgeon to the neurologist, and very properly, too, as the nervous phenomena constitute the most important part of the subject. During this lapse of a quarter of a century neuro-pathology may almost be said to have been discovered. Too much importance, therefore, should not be attached to the older opinions which were formulated by men who, however well informed they may have been in their day, were mere tyros to the specialists of the present time. Physicians will scarcely need this reminder; but it should be timely to lawyers, whose professional reverence for authority and precedent is apt to render them inappreciative of the difference in this respect between science and law.

Existing medical knowledge will not permit us to say that there is any one disease of the human body that cannot possibly be caused by injury, except, possibly, one of an infectious nature. It is quite possible, to be sure, to compile from the medical journals and from one's own personal records a list of the diseases which have been so far caused by injuries, but each new day may add to this list. Whenever an injury has been received, and a disease has followed it after such a length of time as to render it probable that the latter has been the effect and the former the cause, a new observation will have been made. Predisposition—whether hereditary, or from previous

disease, or from coexisting diseases—may render the human being more liable to the excitation of disease by an injury, and it may then become a question as to the respective responsibility of the injury and the predisposition in the production of the disease. Observation, however, has shown that injuries are peculiarly liable to produce certain diseases that have been well studied, and when the symptoms of these are described by some individual whose means of information have not been such as to have made him acquainted with medical writings, their medico-legal value may become so great as peremptorily to exclude simulation. The diseases that can be caused by traumatism are—

Injuries of the bones, ligaments, muscles, and peripheral nerves;
Ordinary myelitis, with secondary degeneration of the motor and sensory tracts;
Locomotor ataxia;
Myelitis of the anterior cornua of the spinal cord;
Progressive muscular atrophy;
Tumors of the spinal cord;
Insanity;
Encephalitis;
Tumors of the brain;
Intracranial hemorrhage;
Chorea;
Epilepsy;
Neuralgia;
Hysteria;
Neurasthenia.

Injuries of the bones are not usually difficult to detect. Erichsen has stated that the brain and cord are not usually injured when the skull or the vertebræ are fractured, because the force of the blow is spent upon the bones and does not reach the underlying nervous tissues. This is a very erroneous statement, both as a matter of physics and clinical observation, and I have been surprised that so many writers should have thought it worthy of refutation by the recital of individual cases. Every hospital *interne* has seen fracture of the skull associated with serious injury to the intracranial contents, and the frequency with which fracture of the vertebræ is conjoined with injury of the cord is well known to every surgeon of experience, and is proven by the cases collected by Thorburn, whilst I can myself cite many from my case-book in refutation of Erichsen's dictum. We should be careful, therefore, not to overlook a nervous injury when we have discovered a bony one. Injuries to the ligaments are usually easy of diagnosis, too, although time and observation may sometimes be needed to exclude an implication of the peripheral nerves. In examining injured muscles myositis and beginning progressive muscular atrophy should be carefully sought for; but it should be remembered that in the latter widespread atrophy of bilateral muscular groups is something entirely different from the wasting of a single injured muscle or sets of muscles. When the peripheral nerves are injured, neuralgia or neuritis will be produced,

for the diagnostic symptoms of which reference must be had to the chapters on these subjects.

Injury of the spinal cord may cause that form of myelitis which is known as ordinary, transverse, or central myelitis (*vide* section on "Myelitis") to distinguish it from the other forms to which special names have been given; and secondary degenerations of the motor and sensory tracts may follow. We know very little at the present time, as has been stated elsewhere, of the symptoms of the secondary degenerations of the sensory columns of the spinal cord; but the symptoms of a secondary motor degeneration are contracture and exaggerated reflexes of the different tendons, such as the patellar reflex, the foot-clonus, hasty micturition, the commingled contracture and exaggerated tendon-reflexes producing the spastic gait. (*Vide* pages 166 and 169.)

It is a certain that locomotor ataxia can be caused by injury, especially by one of a general nature. One of the most marked cases of this disease that I have ever seen quickly followed the celebrated Ashtabula railroad accident, when a train was precipitated from a bridge at a height of three hundred feet.

Myelitis of the anterior horns of the spinal cord, the poliomyelitis of the Germans, or the so-called essential infantile paralysis of the old writers, can be caused by injury.

Progressive muscular atrophy is frequently caused by injury.

Tumors of the spinal cord are also caused by injury, although rarely; and Leyden narrates a case of large tumor of the cord, of traumatic origin, the sufferer from which had been thought to be a malingerer.

There is abundant evidence to prove that injury may cause insanity. The symptoms of mental disease thus produced are not usually well marked, although there is a strong tendency toward dementia. Mania, melancholia, primary dementia, transitory mania, delirium grave, and katatonia cannot be classed among the traumatic insanities, so far as we yet know, but post epileptic insanity can. The different forms of encephalitis that are classed under the generic head of general paresis (see under "Paralytic Dementia") may also be of traumatic origin.

Tumors of the brain, intracranial hemorrhage, and chorea can be caused by injury.

As we have seen (*vide* "Epilepsy"), epilepsy is a symptom of various diseases of the central and peripheral nervous structures, and may also be idiopathic—*i. e.*, caused by cerebral disease of a functional nature; and all these varieties, except the idiopathic, may be caused by injury. Careful regard should be had in this connection, however, to the factors of diagnosis thas have been detailed on page 406.

The relation of hysteria and neurasthenia to injury is the vexed question of medico-legal medicine, and has given rise to most of the modern literature upon the subject. Inasmuch as vast interests of money, life, and happiness are at stake in this query, it is best that we should enter into its merits at some length. Hysteria is a dis-

ease (page 437) that is characterized by certain emotional conditions, peculiar convulsions and paralyses, and by hemianæsthesia. It occurs in males as well as in females, although more frequently in the latter, and also in children. The emotional condition does not necessarily precede or accompany the graver convulsive, paralytic, or hemianæsthetic phenomena. The convulsions are readily distinguishable from those of epilepsy, and even when conjoined with the latter disease, as in hystero-epilepsy, they have marked features of their own, and are of much more favorable prognosis. Charcot teaches that the sole diagnostic symptom of hysteria may be hemianæsthesia, consisting of one-sided impairment of the senses of touch, temperature, pain, and heat, with impairment of the color-sense and concentric limitation of the field of vision; and around this dictum of Charcot's has been waged a furious war. The great French teacher has shown to his classes cases of paralysis involving the arm or leg, with a peculiar limitation of the anæsthetic area, and has claimed that they were hysterical in their nature because the patients also had hemianæsthesia. Oppenheim and Thomsen have found, however, that hemianæsthesia may exist in epilepsy, alcoholism, nervousness, neurasthenia, chorea, conditions of fright, multiple sclerosis, Westphal's neurosis, organic brain disease, and after certain insanities, and that it is not characteristic of hysteria, even in which disease it is very variable, bearing no certain relationship to the bilateral concentric limitation of the field of vision, the most constant symptom of hysteria. Charcot's claim that the functional nervous symptoms of all cases from railway injury are due to hysteria has not therefore been proven. This much may be regarded as settled. Charcot has furthermore been able to produce a paralysis by hypnotism in these so-called hysterical patients, and also to relieve in them an existing paralysis, although the relief has been only of short duration, and he would have us call this paralysis a paralysis of idea, at the same time that he lays great stress upon the distinction between it and an imaginary or simulated paralysis; in other words, as hysteria is a genuine disease of the nervous system, an hysterical paralysis should not be mistaken for one of shamming, although it may closely resemble the latter; but as he does not claim that the same phenomena cannot be produced in non-hysterical individuals, rather relying upon the hemianæsthesia as the basic criterion, it is evident that this point is also not proven. His cases were taken from that great hotbed of nervous disease, the immense hospital of La Salpêtrière, and two of the most remarkable individuals had been respectively thirty and forty years in the institution. Putnam and Walton have reported cases of hemianæsthesia following railroad injuries, and Oppenheim's latest monograph contains a history of traumatic nervous symptoms in 33 cases observed in the nervous clinic of the Charité Hospital of Berlin in a period of five years. That injuries, especially those received in railway accidents, can produce a certain set of symptoms worthy of being considered as a distinct symptom-group was first recognized by Erichsen, and this fact has since been confirmed by many observers. To this symptom-

group Erichsen gave the name of *spinal concussion;* then it was designated *railway brain*, and finally Oppenheim suggested for it the cognomen of *traumatic neurosis*, whilst Charcot, as we have seen, would call it *hysteria*. The symptoms consist of disturbance in varying degree of the various functions of the body, mainly of the nervous system, and the latter are largely sensory and but slightly motor. The sensory symptoms are disturbance of the different senses, such as touch, the muscular sense, heat, pain, sight, hearing, seldom taste or smell; whilst the motor disturbances are expressed by exaggerated tendon and sensory reflexes, and paralysis varying from slight to severe; and there are almost invariably superadded such general nervous disturbances as vertigo, insomnia, a depressed mental condition, irritability of temper, impotence, and loss of appetite. Hemianæsthesia is occasionally associated with these symptoms, as we have already seen. To go more into detail, the course of the symptoms is generally as follows: A man is injured in a railway accident, rendered unconscious perhaps, or perhaps only made dazed and tremulous. He goes home and returns to his work. In the course of a few days or a week he finds that he is not regaining his normal condition of health, that he is nervous, cannot sleep, is not capable of doing his usual mental or physical work, is irritable and generally unstrung. As time goes on, his condition grows worse and he becomes depressed and emotional, easily excited to laughter or to tears, moody, brooding, and showing less and less capacity to apply himself to his usual pursuits and more and more inclined to isolation. If he is examined several weeks or months after the accident, it will usually be found that his tendon-reflexes are exaggerated, perhaps also his sensory reflexes; that there is some impairment of the various senses, or even hemianæsthesia; that his muscles are abnormally weak; that he complains of vague pains, especially about the back, hips, and groins; that his countenance is pale, anxious, and careworn; that he presents the picture, in a word, of a worried and anxious individual; that he has also occasional vertigo, and a sense of distention and fulness about the head, especially at the vertex. This is, I think, a fair general picture of the symptoms in this class of cases—in other words, of the type; but there are many variations from this type, so that in one case one set of symptoms may predominate, and in another case a somewhat different set may be presented. It has been claimed that neuro-retinitis has been occasionally observed, but I have never seen this myself, although it has been my habit to examine carefully every case with the ophthalmoscope. The attitude, the movements in sitting down and rising, the peculiar efforts to effect a voluntary movement, and the gait, are all characteristic, as Oppenheim first had the acumen to observe, although I have myself noted the same thing in many cases without having it occur to me that there was anything distinctive about them. The body is held anxiously fixed, the spine bent stiffly forward, in sitting down or rising the patient seeks support with the hands, and he changes his attitude carefully and slowly. Oppenheim attributes this mainly to the rigidity of the dorsal muscles, and only in less degree to in-

stinctive avoidance of movements of the vertebral column, and I think his explanation is thoroughly accurate. In making a voluntary movement the patient seemingly makes a great effort, and yet the resulting muscular movement is relatively feeble, so that the impression upon the observer is that of simulation on the part of the patient. I must confess that such was my belief in my days of less experience, although further observation has firmly convinced me that such a conviction does grave injustice. Oppenheim attributes this muscular weakness to the fact that the motor impulses coming from the brain are not so precisely directed as in health, so that they do not pass in full volume to the muscles for which they are intended, but are erratically diverted to other muscular groups subserving other but allied movements. Thus, if the patient is told to press the observer's hand with his full strength, the shoulder-muscles, the flexors of the elbow, and the extensors of the hand are simultaneously thrown into powerful action. When muscular paralysis occurs, it is noteworthy that it is seldom or never confined to muscular groups supplied by one nerve-trunk. The motor paralysis is generally a flaccid one, although it may be conjoined with contracture, which may be due to the pain that is experienced in certain muscular movements, or which may be truly hysterical. The gait is peculiar. The patient walks with legs wide apart, with short steps, and slowly; the movements at the joints are limited, the foot being drawn along with the whole plantar surface, or the heel shuffling on the floor, whilst the body is held in the position just described. Usually one hand is placed on the back or on the side. Although this gait is like that which is usually known as spastic, it yet differs from it in that the toes do not cling to the floor, and in that there is not the springiness as of a stiff bedspring (page 276). Various modifications of this gait may be observed, but they can always be traced, in my experience, to the same causes at play in different muscles, viz., pain, giving rise reflexly to pseudo-contracture. If all these symptoms are analyzed, no other conclusion can be reached than that they are the effect of exaggerated reflex action.

We do not know what is the molecular condition of the great nervous centres causing this exaggerated reflex action. Westphal has advanced the theory that the cause is to be found in small foci of myelitis or encephalitis, analogous in their action to multiple sclerosis. Bramwell suggests that there may be multiple capillary hemorrhages in the brain and cord, giving rise to inflammatory processes, and eventually to sclerosis; and Vibert found in those killed at the great railway accident at Charenton in 1881 very abundant punctate hemorrhages in the upper part of the body. Willigk has detected dilatation of the finest vessels, with infiltration into the perivascular spaces and degeneration of the vascular coats. Mendel fastened dogs on a table revolving 125 to 130 times a minute, the revolutions continuing for half an hour, the heads of the animals being toward the periphery, and thus produced punctate hemorrhages in the brain and cord. One hundred and ten revolutions for six minutes a day, continued for several weeks, caused adhesions between the skull and the brain

and its membranes, an increase in the nuclei in the cells of the connective tissue, as well as in the number of vessels, and changes in the nerve-cells. Fürstner, repeating Mendel's experiments, with sixty to eighty revolutions for one or two minutes, found double primary degeneration of the lateral columns and of part of the posterior columns, alteration of the optic nerves, and cerebral change similar to that described by Mendel. Dumésnil, Petel, and Edes have reported degeneration of the lateral columns in the human being. But it should be observed that Westphal and Bramwell only theorize, that Vibert speaks alone of individuals who were killed by an accident severe enough to cause quick death, that Willigk simply mentions one individual, that Dumésnil, Petel, and Edes report no more than a few cases, and that Mendel and Fürstner obtained their results by a method which, however ingenious, is not at all analogous to ordinary injuries or those produced by railway accidents. I may, therefore, repeat my assertion that we do not know the molecular cause in the central nervous system that produces these exaggerated reflexes. Clevenger enters into an ingenious argument to demonstrate that they are due to injury of the sympathetic nervous system ; but, even if we admit that this is correct, it only transfers the mystery from one nervous system to another, whilst I do not think that the marked sensory and motor symptoms can be at all explained by any perverted action of the sympathetic. It has been suggested that spinal concussion is the cause, but this suggestion can only be due to confusion of thought, because a concussion is simply another name for an injury, and it is the molecular effect of the concussion or injury that can alone give rise to the clinical phenomena. Arguments about the question whether spinal concussion can exist are as idle as the endless discussions of the schoolmen. In the present state of our knowledge we cannot do better than to say that these symptoms are functional—*i. e.*, due to molecular alterations in the brain and cord that have not been detected by our present methods of preparation and staining of nervous tissue, even if it is possible that such methods can detect them.

Belonging to the functional nervous affections, therefore, as this particular group of symptoms undoubtedly does, I think that we should classify it with those other groups of symptoms which are known by the name of neurasthenia ; and, instead of adding to the confusion already existing in our medical nomenclature by coining such new names as spinal concussion, railway brain or spine, or traumatic neurosis, I think that we shall greatly simplify matters by simply designating it as *traumatic neurasthenia.* We have already seen that there is no warrant for regarding this traumatic neurasthenia as hysterical. It undoubtedly possesses a certain likeness to other forms of neurasthenia so far as regards the paræsthesia and the general nervous and mental symptoms ; but it differs from them in the painful sensations, the muscular rigidity, the peculiar attitude, the gait, the disturbed muscular innervation, and, finally, in the prognosis. There has been much difference of opinion in regard to this very question of prognosis, but I think that this has arisen mainly from the fact that sufficient regard

has not been paid to the peculiar circumstances in which patients of this class are usually placed. Few observers will deny, I think, that the symptoms of traumatic neurasthenia, especially those due to railway accidents, almost invariably continue for a long time, disabling the patients for many years, and in some cases even terminating fatally; but some of these very observers contend that the maintenance of the symptoms is due to the psychical condition of the patient, whilst others insist that it is inherent to the disease. Certain it is that the psychical condition of these patients is a very unfortunate one. However uneducated they may be, newspapers and the talk of every-day life has filled their mind with dread of the mysterious and baleful consequences that may happen to those who receive injuries, particularly in railway accidents. They have also heard for years of the damages, often enormous, which corporations have been obliged to pay. When the accident occurs, the nervous system undoubtedly receives a shock, perhaps intensified by the sight of the killed and wounded, with all the attendant horrors, and this shock should receive immediate and judicious treatment by rest, isolation, and medicaments; but instead of this, a lawyer or his agent—the so-called "runner" of this country—quickly appears upon the scene, and spurs the patient on to a suit for damages by exaggerating the injury and its consequences, so as to make the too-willing sufferer believe that the company can be readily forced to pay damages. Then come the long years of weary suffering, anxiety, waiting, and disappointment, unrelieved by proper treatment, for although the patient and the lawyer may not consciously discourage treatment, yet too many hopes and interests would be blasted by a cure to permit of treatment ever being properly carried on, even if any self-respecting physician could be found to undertake it. Months, perhaps a year or more, are passed in waiting for the suit to be tried. I have known of three years having elapsed before this was done, because of delay in obtaining the evidence, then in fixing the responsibility upon the right corporation, then by alleged corruption of the attorneys, and, finally, by the long waiting before the case could be reached upon the calendar of the court. When the suit has been brought and pushed to a successful termination, an appeal to a higher court will usually be taken, perhaps to a second higher court, and thus months or years more pass by. In some cases it may even happen that a successful issue in the court of the highest resort is contested upon a charge of conspiracy or some matter of legal technicality that is concocted in order to gain time. Finally, the case being at last successfully ended, it may turn out, much more frequently than is dreamed of by those who have not had a long experience, that the costs of the action and the lawyer's fee will leave but a pitiable sum of money at the disposal of the patient. This is not an exaggerated picture by any means. It is a composite portrait of dozens of cases whose details can be taken from my case-books. All this disturbance that follows the accident is oftentimes, I am firmly convinced, a more potent cause of the neurasthenia than the accident itself, and I feel sure

that it is this secondary psychical disturbance that makes this form of neurasthenia more intractable than other forms of neurasthenia, although I cannot quite agree with the statement which I have often heard made on the witness-stand by very competent colleagues, that traumatic neurasthenia does not differ from other forms of neurasthenia except in the psychical element. Notwithstanding that Charcot does not exactly say this, it is yet evident that he takes very much the same view in regard to the hysterical origin of these cases. Not only, however, are their symptoms to a certain extent pathognomonic, but there never yet has been any clinical proof adduced to demonstrate that the difference between them and other neurasthenic types is due alone to the mental element. Nevertheless, I firmly believe that it is the psychical element, together with the resulting lack of proper treatment, that makes the usual unfavorable prognosis of this class of cases. In several instances I have persuaded patients to place themselves promptly under treatment, and at the same time either to abandon or compromise the legal proceedings, and in every instance a cure has been effected. In other words, in order to make my meaning perfectly plain in a matter of this importance, let me repeat by stating that while I do believe that traumatic neurasthenia is a species of disease that has distinctive features of its own, I do not believe that it is of unfavorable prognosis, provided that the psychical element can be excluded and that prompt and proper treatment can be undertaken. It must be clearly borne in mind, however, that this statement applies only to this group of functional symptoms, and that it does not apply to the organic diseases caused by injury; but moral responsibility cannot be hereby avoided by the persons or corporations by whom the injury has been caused, for whether the neurasthenia is rendered more intractable or not by the accompanying psychical element, the injury itself has been the cause of all the symptoms. Whether the legal responsibility of these individuals or corporations is lessened on this account is a question of law for the courts to determine, and we physicians have nothing whatever to do with that aspect of the question.

The element of simulation in all these diseases produced by injury should always be carefully considered whenever there is a question of a suit for damages. Physicians make a great mistake, however, in entering upon the examination in a mental state of bias against the alleged sufferer, and justice will be much more equally done if all the symptoms are carefully and impartially gathered before any conclusion is reached; but a sharp distinction should always be made between the symptoms that are *objective* and those that are *subjective*—*i. e.*, between those for which we must entirely or mainly rely upon the patient's statement, attitude, or facial expression, and those which we can entirely or absolutely determine for ourselves. Nevertheless, the value is very great of a group of symptoms that are usual to a disease, even if many of these are so purely subjective as to make us dependent in a large degree upon the patient's statements. For instance, the stabbing and lancinating

pains of locomotor ataxia cannot be seen or tested by the examining physician, and yet an accurate description of them by a patient should carry conviction, unless there is reason to believe, as will very rarely happen, that the patient has special sources of information; and this is also true, in varying degree, of the cincture feeling and bladder and rectal symptoms of myelitis, the slighter losses of consciousness of epilepsy, and the paræsthesia, vertigo, nervousness, insomnia, and peculiar emotional condition of traumatic neurasthenia. Nor should a purely subjective symptom lose one iota of the value that it ought to have when it is conjoined with demonstrable objective symptoms in such a way as to picture properly a well-known disease. Thus, when we can see with our own eyes that a patient has the glossy skin, the tapering and slightly swollen fingers, the muscular wasting, and the altered electrical reactions of an ulnar neuritis, we should not reject the statement of pain, numbness, and tingling which may be described. The courts have a very vicious habit of ruling out the statement of a physician that the symptoms, when taken together, indicate a certain disease, and of forcing the medical witness to confine himself to an enumeration of the individual symptoms, leaving the jury to be the diagnosticians.

Subjective symptoms are—

The senses of touch, pain, temperature, and the muscular sense;
Paræsthesia;
Hearing;
Sight;
Smell;
Taste;
Urination and defecation;
Sexual sense;
Vertigo;
Losses of consciousness, with or without convulsions;
Insomnia;
Nervousness;
The mental condition.

Whilst objective symptoms are—

Paralysis;
Exaggerated tendon and sensory reflexes;
Contracture;
Ataxia;
Muscular atrophy and hypertrophy;
The attitude and gait;
The ocular and retinal condition;
Œdema and swelling;
Fractures and dislocations;
The facial expression.

Whilst many of these symptoms have been considered in Chapter II. (pages 154–174), also in connection with the diseases in which they have been found, and whilst others are so very familiar to the physician as to need no more than a bare mention, it is necessary to go into special detail about the medico-legal bearings of most of them.

In testing the sense of touch the ordinary method of employing rough and smooth objects and an æsthesiometer will not exclude simulation. The test by means of a pin or other penetrating object is not a test of the sense of touch, as it has been assumed to be in so many trials, but really of the sense of pain; and this is also true of the test by means of the powerful faradic current. The best medico-legal test of the sense of touch known to me is to touch lightly the patient upon the alleged anæsthetic area when his attention is diverted or when his back is turned, due attention being paid to differences in tactile sensibility normally existing in the different areas of the skin, and to any possible marked degree of mental dulness. This simple test is extremely effective, and I have never known it to fail, even with the best informed malingerers, and it is especially useful in alleged hyperæsthesia. The best test for the sense of pain is a powerful faradic current. A very gentle current should first be applied, and should be gradually and carefully increased to a moderate degree, so as not to alarm the patient, and then, the physician's hand having been all the time kept on the current-regulator, a powerful current should be suddenly turned on. Some means of measuring the strength of this current should be employed, so as to be able to reproduce it and allow the jury, if necessary, to test it for themselves. The strength of the current that is to be used should always be determined before the examination, so that care may be exercised to employ no more than will be necessary to cause a distinct and not extremely painful sensation. The test by means of a needle is extremely unreliable, even though that needle be concealed in the so-called *aiguë caché*, or hidden needle, for we must remember that hypodermic needles can often be thrust for inches into the tissues without exciting pain, so that it is a very easy matter for any determined person to accustom himself to bury a pin in his flesh, as is often done in jest by boys. The exact position of the alleged pain and the attitude and gait are frequently of great importance. If the patient marks out the pain in the distribution of a nerve, this is strong confirmation of the truth of his statement, although the converse of this proposition does not follow, for pain is often vaguely distributed in indubitable disease; so that the positive value of such a statement is great, whilst it has no negative worth. The presence of the characteristic attitude and gait of sciatica and of traumatic neurasthenia would be of great value. A very efficient method of testing the temperature-sense is as follows: The patient should be blindfolded; a tumbler or test-tube should be filled with cold water, a second with moderately warm water, and a third with water so warm as to be slightly painful but not to scald. The glasses containing the cold and the lukewarm water should then be applied in succession to the portion of the skin to be tested, and the patient be asked to state which is the cold and which is the lukewarm. This should be repeated several times, when, if the patient has failed to distinguish the temperature of the water in the two glasses, some of the very hot water should be suddenly spilled over the alleged thermo-anæsthetic area, care

being taken not to spill any water upon any portion of the skin that has not been alleged to be affected. I know of no test of the muscular sense that will positively exclude simulation. When a patient alleges that the sense of pain, the muscular sense, or the temperature-sense is alone affected, whilst no claim is made as to any implication of the sense of touch, this would argue strongly against simulation; for any malingerer, except one versed in nervous disease, would be most likely to insist upon impairment of the sense of touch. It should also be borne in mind that these four different senses may be affected in different diseases, either singly or in conjunction with one, two, or all of the others.

PART III.

MENTAL DISEASES.

CHAPTER I.

CLASSIFICATION OF INSANITY.

It may be justly regarded as an axiom that whenever great stress is laid upon the classification of diseases the knowledge of those diseases is as yet in its infancy. In the older text-books upon insanity, therefore, much more attention was paid to the classification than to the pathology and the clinical symptoms, and in many of the recent ones this ill-omened heirloom of earlier times has been thought to be a matter of great importance. In reality the classification of the insanities is best made when the different types are simply catalogued. Of what earthly consequence is the classification of the different forms of disease that we have enumerated in the earlier portions of this book, such as locomotor ataxia, Huntington's chorea, acromegaly, myxœdema, etc.? and who would think of troubling himself as to whether locomotor ataxia should be classified among the spinal diseases, or the cerebro-spinal, or the peripheral, when in reality it may be any one of the three? In the same way, what possible difference does it make under what heading we put paranoia, melancholia, mania, delirium grave, or general paresis? The classification of insanity, therefore, that I should adopt, would be the simple enumeration of the different types, and the subtypes of each one of these. My classification, for the purposes at least of the practising physician, is therefore contained in the table of contents. I would like to impress upon the readers of this book the folly of attempting a classification by mere symptoms, inasmuch as the same symptom may obtain in different types of insanity just as the same symptom may be observed in different types of all other diseases.

CHAPTER II.

DELUSIONS, HALLUCINATIONS, AND ILLUSIONS.[1]

THERE are three symptoms of the insane with which the general practitioner should be familiar, namely, delusions, hallucinations, and illusions.

A delusion is a false belief. It has sometimes been said that the delusion or false belief of the insane differs from the false belief of the sane in the fact that the former is fixed and not amenable to reason. This is a mistake. The delusions of the insane are often not at all fixed, but fleeting, and in a certain degree amenable to reason, whilst the false beliefs of the sane are often fixed and not at all amenable to reason. Indeed, what can be more fixed and less amenable to reason than the beliefs of sane people about creeds, politics, religion, matters of science, theology, etc.? The only definition of an insane delusion is that it is a false belief occurring in an insane person. Whether or not a person is insane is a question of diagnosis.

An hallucination is an imperfect perception through any one of the different senses, and therefore hallucinations may be as various as are the senses; so that, as there are the different senses of smell, sight, taste, hearing, touch, pain, temperature, muscular sense, the sexual sensations, and the visceral sensations, we may have hallucinations of all these. Thus, a person who imagines that he sees something, or hears something, or tastes something, or feels something that he is not seeing, hearing, tasting, or feeling, has an hallucination.

An illusion is a conversion of something really perceived into something that is not perceived. For instance, if one sees a table before him and imagines it to be an elephant, or hears a street-car and imagines it to be a cannon, he converts something real, the table or street-car, into something unreal, the elephant or cannon, and he has an illusion.

If these hallucinations or illusions are believed in, they are delusive; if not believed in, they are non-delusive; so that we can have delusive and non-delusive hallucinations and illusions.

It has been shown that many forms of insanity have always the same kind of delusion, so that it becomes one of the diagnostic symptoms, and these have been called by Griesinger *primordial delusions.* It is extremely probable that the future will show that each form of insanity has its primordial delusion, if it has any at all. Thus, it is known at the present day that the primordial delu-

[1] Delusion is derived from *deludere,* to cheat; hallucination, from *alucinari,* to wander in mind; and illusion, from *illudere,* to play with anything; so it is very evident that the modern technical meaning attached to these terms is not explained by their etymology.

sion of paretic dementia is one of grandeur; of paranoia, a commingled delusion of persecution and self-exaltation; and of alcoholic insanity, marital infidelity. Many forms of insanity have also a characteristic or primordial hallucination, so that in mania the hallucinations are genial and exhilarating; in melancholia, terrifying and depressing; whilst in alcoholic insanity the hallucinations are mainly auditory, often startlingly so.

Illusions have not been so well studied as hallucinations, because they are much more difficult of detection clinically. If, for example, a patient speaks of having heard a cannon, it may be very difficult to ascertain that he has simply heard a street-car and supposed it to be a cannon; or if he complains of having seen an elephant, it may be difficult to know that he has merely seen a table and imagined it to be an elephant. Several years ago I was concerned in a will case in which several days were wasted trying to prove that the testator had had illusions of smell. He had imagined that a certain closet in his house had an offensive odor. None of the family or servants could detect it. A plumber was sent for, but he could perceive none. The Board of Health was notified, sent two inspectors, and they could smell nothing. It was also shown that there were no pipes in the closet or anywhere in the adjoining wall or floors. The floor, however, was taken up, and the lath and plaster of the walls were removed, and nobody but the testator during all this period could detect any odor. Yet the counsel upon the opposite side laughed the whole matter to scorn, and contended simply that the old man was nervous. The testimony of a rhinologist was introduced to show that the testator had had no nasal disease capable of causing the odor. And yet the will of this old lunatic was admitted to probate!

The question whether delusions and hallucinations are by themselves evidence of insanity has been much discussed. There is no doubt, however, that they can both occur in persons who are sane. Hallucinations especially are common with those whose imagination is artifically cultivated, as in poets and novelists. Dickens is said to have wept over the death of Little Nell, and was able to conjure up the different characters in his novels to his mind's eye as vividly as if they had been existant. Burns could never have written that vivid account of the witches' orgie, in "Tam o'Shanter," without a mental process that is identical with what we call an hallucination. When Shakespeare causes Macbeth to fall back affrighted at the vision of the murdered Banquo, an hallucination of sight was pictured such as might readily have occurred in that age to an overwrought mind. No great work of art, no great poem, no great drama, no great work of fiction, can be written without an hallucinatory process going on in the mind of the writer throughout the work of composition. Milton's *Paradise Lost* and Dante's *Divina Commedia* are simply a series of magnificent hallucinations of a healthy, great mind. A most remarkable instance of a similar kind is contained in the story of Goethe encountering a vision of himself as he rode to Rosenheim. Because Constantine the Great saw the vision of the Cross, because

Mahomet saw the heavens of the future Koran opening before him, and because other enthusiasts have had hallucinations of sight and hearing, it does not follow that they were insane. Indeed, hallucinations can occur in some conditions of simple overstrain, neurasthenia, and hysteria, although it must be admitted that they are rare, and that their continuance would be very ominous. It is equally certain that delusion, accompanied or not by hallucinations, may occur in the sane, and of this we have confident proof in the belief of the Highlanders of Scotland in the so-called second sight; *i. e.*, the faculty of seeing upon the deathbed people who are destined to die; as well as in the widespread belief in this country in the so-called Spiritualism. Every reader of this book is probably acquainted with perfectly sane people who are firm believers in the hallucinations of hearing and sight of Spiritualism. Hallucinations and delusions, therefore, are like other signs of disease—their value must be determined by accompanying symptoms. An hallucination or delusion in a person who otherwise presents no evidence of lack of mental health would be of no importance unless there was a continuance of it. Medico-legally, delusions and hallucinations often become difficult to prove because of the ignorance of juries and lawyers regarding medical science. In one case of mine, for example, it was shown that a certain individual, who was on board a steamer plying between New York and Cuba, came up to the captain when they were upward of three hundred miles out in the Atlantic Ocean, and called his attention to something that he thought he saw afar on the water. The captain could see nothing, but my patient was so persistent that the captain got his glass and scanned the horizon carefully. Still he could see nothing. Then my patient broke out in a tone of anger: "Don't you see those horses, and that brougham, and John," who was his coachman, "sinking there into the water?" Although all this occurred, as I have said, three hundred miles out on the Atlantic Ocean, an expert was found who was willing to testify that this was not an hallucination, nothwithstanding that there were many other symptoms of mental impairment. Certainly, it is not possible for human testimony to produce any more conclusive evidence of an hallucination than was here afforded.

CHAPTER III.

MORBID FEARS AND IMPULSES.

THERE are certain morbid fears or conceptions, what the Germans call *Zwangsvorstellungen*, and certain morbid impulses, which they call *Zwangshandlungen*, or imperative acts, that are sometimes classified as neurasthenia, as has been done by the late George M. Beard, as well as by his disciples in the posthumous literature which he originated in foreign countries. These are in exceptional cases conjoined with insanity, but they are better considered, I think, under the simple heading of morbid fears and impulses. True neurasthenia should be limited to the forms which I have referred to before (*vide* section on "Neurasthenia"); and as most of these cases of morbid fears and impulses present no other mental symptoms, it is not warrantable to class them as mental diseases. The most common morbid fears are: fear of places or localities, and fear of pollution. Fear of places was first described by Westphal under the name of *agoraphobia*. Gélineau calls it *kenophobia*. The name of *claustrophobia* has been given by Verger, of Milan, to a morbid fear of closed places. These individuals have a great dread of certain places, sometimes of a large place or hall, sometimes of a bridge, sometimes of a ferryboat, etc. A patient of mine came to me one day, with horror depicted upon his face, to state that he could not go over the Brooklyn Bridge, or, indeed, over any bridge at all, or in any of the ferryboats. After talking with him in vain, and ascertaining in a quiet way that he was utterly unable to control his morbid fear, I simply told him to come to me on the following Sunday, only two days off, and I would treat him. When he came I had my carriage ready, got into it with him, and drove to the Brooklyn Bridge. There I got out, took his arm, and told him I proposed to walk across with him. I do not think I have ever seen a more marked picture of mental agony than was presented by this man, who almost sobbed as he begged me to desist. I persisted, however, and walked across with him. Then I turned around and walked back with him with much more ease. I then drove to one of the ferries, got out, took my patient again by the arm, and crossed with him, having a milder repetition of the same mental agony; and after reaching the other side came back on the same boat. I then said to him that he could now see how groundless his fears were, and that he ought to cross alone, which he did. I also advised him to make it his business to cross the bridge and ferries two or three times every day, and to go out of his way to cross any bridge that he knew of, and by this and other means (of which I shall speak when I come to the treatment) he was cured completely. These cases

must be distinguished from the vertiginous sensations which some people afflicted with lithæmia or aural disease or refractive errors experience in similar places. The fear of pollution was first described by LeGrande du Saulle, under the name of *folly of doubt,* and later by William M. Hammond under the name of *mysophobia.* Some authors have made a distinction between Du Saulle's cases and those of Hammond; but I think they are both pseudo-insanities of doubt, varying simply in the clinical manifestations, and I therefore believe that the name given by LeGrande du Saulle is the best one—insanity of doubt, or *folie de doute.* These patients have a morbid doubt about everything that they do. One case of mine would go out of a door, close it, and then come back, uncertain as to whether he had closed it, close it again, go off a little way, again feel uncertain as to whether he had closed it properly, go back again, and so on for many times. Another patient washed her hands innumerable times in the day, uncertain as to whether she had really got them clean, and it was a case of this kind which caused Hammond to coin the name of mysophobia, or fear of pollution, as his patient claimed that she had such a fear.

Morbid impulses are mainly acts which grow out of morbid fears, and it is not necessary to go into them at length, as they are largely treated of in the chapters upon sexual perversion and the different insanities. There are many which are scarcely worthy of classification. Thus there is an impulse to set fire to things (pyromania); an impulse to collect pins; an impulse to collect figures (arithmomania); an impulse that Magnan has observed in certain old maids who have a characteristic love for cats, and to which he gives the magnificent name of *folie des anti-vivisectionistes;* whilst morbid impulses to jump from great heights are very common, those of pregnant women are innumerable, and the ones which may come to different persons at various times are almost uncountable. Most of these individuals have a neurotic predisposition, either personal or hereditary, but it is exceptional to find in them any evidences of mental disease. The medico-legal responsibility of these individuals is a vexed question. Of course, it is possible, as we have seen, that these morbid fears may be so far beyond volitional control as to render the person irresponsible; but of this clear legal proof should be adduced, because it is too often that a morbid impulse of this kind is feigned to cloak an understood motive. Thus, in one case in which I acted as expert on behalf of a county not far from New York, a young girl had set fire to her dwelling-house in such a dangerous manner that if the flame had not been quickly discovered it would probably have burned the building to the ground, together with several adjoining ones. It was alleged that she was suffering from menstrual insanity, and that her prominent symptom was pyromania. There was not a single symptom of insanity adduced, however, either before or after the act. On the other hand, it was clearly shown that this girl, who was extremely pretty, had fallen in love with a certain young man in the city of New York; that she was in the habit of meeting and going to houses of assignation with him; that

she had her goods insured for a sum which, though small, was large to her; and finally she confessed that she had set fire to her building in order to obtain enough money to go to New York and live with this young man. The jury, sapient as American juries are, and as juries probably are all over the world, found that she was insane; but I was told that their real reason in bringing in this verdict was that they thought the girl had already been punished enough; that the confession had been wormed out of her by the arts of a professional detective; and that the judge who was trying the case was so famous for his severe sentences that it was very probable he would sentence her to a year or more of imprisonment. So they took the matter into their own hands. This was all very fine, of course, from an emotional standpoint, but it was nevertheless a travesty on science.

The prognosis of these cases of morbid fears and impulses is usually excellent, with proper treatment.

The treatment requires considerable tact and judgment. If the patient's general health is in any way depressed, this should be carefully attended to. If the morbid fears and impulses occur at any time of functional disturbance, the disappearance of this will probably be sufficient to relieve them. Thus, cases that occur during menstruation or pregnancy need no special treatment. The young man whose case I have detailed, and who had such a morbid fear of the bridges and ferries, was completely cured by placebos, suggestions, and being made to cross the bridges and ferries. Curiously enough, several years after the time of which I have spoken, he came to me again with a return of his fears; but as these were evidently caused by the impending death of his mother, I talked to him gently and kindly, gave him a placebo, and after his mother had died and he had returned to his normal condition, he had no further trouble. I know of no medication by drugs that is of the least value. In my opinion, suggestion is the best method. If any one of my readers will carefully peruse the section upon "Hypnotism," he will obtain some idea of the vast power of hypnotism, which is probably, as Bernheim claims, mainly psychical suggestion; and this principle of suggestion applied to these morbid fears will often work wonders. In some cases it will be well actually to hypnotize the person. In many, however, it will not be necessary to do this. Let the physician gain the confidence of the patient, talk to him gently and sympathizingly, use a placebo in the way of a drug or some application of electricity or the passage of a uterine or urethral sound, and speak very confidently of the results that will come. Whatever suggestion is made, either with or without hypnotism, it should be offered quietly and unostentatiously. It will sometimes be necessary to take women, especially young women, with or without a tendency to hysteria, away from the environment of well-disposed, sympathizing friends, whose judgment cannot be trusted and who have no conception of the disease, and place them in charge of a trained nurse of tact and judgment. Care must be taken, however, that the nurse should be of kindred tastes and social habits to that of the patient, and the great mistake should never be made of placing

a coarse, illiterate nurse with a refined and educated woman, or one who is a fool with a woman whose intellectual capacity is high. With men, however, aid can scarcely ever be obtained from a nurse, partly because male nurses are not so well adapted for the treatment of nervous diseases as female nurses, and partly because men are not so docile as women. No case of this kind should ever be sent to an asylum unless there are marked evidences of mental disease. Even where there is a tendency to suicide, this can be guarded against quite as well outside of an asylum as in it, because these cases would after a while be allowed almost as much liberty in an asylum as they would have at home. Psychical therapeutics, in a word, is of more value in these cases than visceral or vascular therapeutics.

CHAPTER IV.

SEXUAL PERVERSION.

To enumerate all the varieties of sexual perversion would be to catalogue the depths of intelligent human depravity; but there are certain well-marked types that are so frequently conjoined with mental disease or that are observed so often as to warrant a classification.

Although these sexual perversions may be associated with insanity, their presence is not of itself evidence of the latter; indeed, it may be said that there is no one single symptom that is evidence of mental disease, any more than there is any one single symptom that is evidence of other visceral disease.

The main forms of sexual perversion may be thus enumerated:

Masturbation;
Sodomy;
Anthropophagy;
Necrophilism;
Homosexuality;
Sexual hermaphrodism;
Masochismus;
Sadismus;
Erotic fetichism;
Sexual metamorphosis.

Masturbation is a very common disease with boys, less common with girls. Every large school, especially if it be a boarding-school, is apt to be a hotbed of this sort of thing unless the discipline is very thorough, and children teach one another to indulge in it without the slightest inkling of the baneful effects. The habit, however, is generally discontinued, I think, after adult age, and I should, therefore, be inclined to look upon its persistence then as a sign of true sexual perversion, whereas it might have been formed in a child just as it would learn to walk in a peculiar way, or learn to talk in a peculiar way, or learn to do other things as it sees other people doing them at that imitative age. That the habit is a baneful one there can be no question, but there may well be a doubt whether we know exactly the extent of its banefulness. It is one of the traditions of medicine handed down for hundreds of years that a masturbator can be recognized by his furtive countenance, by his tendency to introspection, and by his *facies*, which, like almost every other *facies*, is indescribable, but is soon recognized with a little experience. I believe that this description is often a good one of the manner of an individual who thinks that his masturbation is a sin, and who therefore feels ashamed of it; but I do not think that the masturbator who is not ashamed of his habit has any such hangdog look. Any human

being who does something that he knows to be wrong, or that he thinks is wrong—whether it is really wrong or not makes very little difference in its effect upon the mind so long as it is thought to be wrong—will have a furtive countenance; but I have very great doubt whether there is anything more peculiar about the countenance of a masturbator than there is about the countenance of a burglar or a smuggler or a counterfeiter or a confidence man. The physical effects of masturbation are not the cause of the manner of the *facies.* There has been so much confusion of thought in this matter that very vague assertions have been made which will not bear analysis. Epilepsy, insanity, neurasthenia, and a host of other ills have been attributed to masturbation. It is possible that the habit may sometimes act as a predisposing or exciting cause of disease by an excessive loss of the seminal fluid if the masturbation be frequently repeated; but I have seen very few instances in which this causal relationship was indicated, and I have never yet seen a case of any disease that has been directly caused in this manner. I do not wish to be understood, however, as saying that it is not a habit extremely deleterious to the general health; but I wish to make it plain that its effects are more largely psychical than physical. The insane are very prone to masturbate. In men this can, of course, be readily detected. In women, however, some skilful observation may be needed to ascertain the fact, and in some cases it will only be indicated by a peculiar swaying of the body, sometimes continued for hours, an adduction of the thighs, and an absent, semi-ecstatic look at times.

Sodomy[1] is a very common form of sexual perversion in our civilized communities, much more so than is dreamed of by the general public. Every hotel superintendent and every police captain or detective can verify this fact, and the individuals suffering from this sexual perversion are a source of great annoyance to the former. I am told by those who are addicted to this vice that they can go at any time into one of the main thoroughfares of a city like New York and be certain of finding one or two fellow-spirits in a distance of a few blocks. They recognize one another by peculiar and indescribable signs and gestures, and when I have endeavored to find out what these were I have been simply told that they cannot be put into words, but that they are such ordinary subtle signs as those by which it is recognized that a woman is inclined to flirt, that she is a street-walker, or that there is something suspicious about her. I have in my possession the diary of a male patient who has now reached middle age, and who has been a sodomist since boyhood. The autobiography is an interesting one, and would read quite as well as the autobiography of that other sexual pervert, Jean Jacques Rousseau. It tells, in words made vivid with delicate shades of emotion, how one spring day he and a congenial companion wandered off to

[1] By sodomy I mean unnatural sexual intercourse between persons of the same sex. Bestiality is the intercourse of human beings with animals. Pæderasty is sodomy with boys. There has been so much confusion in the use of these terms, especially in medical and law works, that these distinctions should be made.

the glades of the Park, how they mutually expressed their love, how from time to time they penetrated deeper into the thickets away from the gaze of man, and how finally, as the stars came out, they loitered home in the twilight, hand-in-hand like Paul and Virginia, exchanging gentle confidences. This individual came to me with the idea that he might get rid of his sexual perversion. He bowed sufficiently to the conventionalities of the world to recognize that it was wise for him to give up the habit, the more especially as an enemy had gotten an inkling of the fact and forced him to abandon a certain scheme. But all the same he derived no pleasure from sexual intercourse in the ordinary way. The pæderasty that is punished in the State of New York was praised in beautiful sonnets by Hafiz, the Persian poet, who sings thrillingly of the charms of young boys, and, indeed, more than one revolution occurred in classic Athens from sexual passions of this kind. These sodomists are divided into female and male, and the one who acts as female always acts as female, while the one who acts as male always acts as male too, for the reason, as they tell me, that the one who has become accustomed to the receptive posture of the female can never derive any pleasure except in this position, and the same is true of the one who takes the aggressive attitude of the male. It has seemed to me, too, that there is a difference mentally between the males and the females of this sodomist world, the males having the virile characteristics of males, whilst the females are more effeminate and gentle. I persuaded one of my sodomistic patients to bring five or six of his fellow-sodomists with him to my office, and I was perfectly amazed to witness the shamelessness with which they answered questions and went into details. I firmly believe that in every large city there is a large number of individuals addicted to sodomy, and who form, as it were, a community apart. I know of no signs by which they can be recognized, and if I were at liberty to-morrow to tell the names of the lifelong sodomists that I know in and around New York, it would constitute a *chronique scandaleuse* of the most sensational kind.

Anthropophagy is that variety of sexual perversion which is accompanied by mutilation and cannabalism. It is probable that the murders of "Jack the Ripper," which have excited so much attention of late years in the Whitechapel district of London, have been done by a sexual pervert of this class, and that the mutilation of the bodies was the regular gratification of a perverted sexual desire. It is said that several of the Cæsars took great pleasure in seeing virgins slaughtered. Some of these individuals will cut out certain organs, as the genitalia or the breasts, and eat them, or drink the blood. Sergeant Bertrand was a celebrated instance of this form of sexual perversion. He would dig up the bodies of young girls, cut them open with a sabre or pocket-knife, tear out the entrails, and then masturbate. After a while he became indifferent to the sex of the corpses, and he next grew accustomed to actual coitus with female bodies.

Necrophilism is that form of sexual perversion in which dead bodies are dug up and violated.

Homosexuality is that form of sexual perversion in which the individual conceives a violent sexual passion for one of the same sex, and gratifies it either by sodomy, by titillation, or platonically. A curious book was written on this subject by a German officer of high judicial standing named Ullrich, in which this form of sexual love was warmly defended, it being claimed that it was justified by natural laws, and he instances its occurrence in certain insects and among certain of the ancient nations. These individuals were called *Urninge.* Cases of homosexuality are by no means infrequent. Only a few years ago an instance of this kind occurred in Memphis, Tennessee. A young girl, Freda Ward, had a female friend to whom she became very much attached. They had first met at school, and after leaving embraced every opportunity of meeting, until attention was attracted to the matter, and they were forbidden to associate with each other. Upon this the sexual pervert murdered her girl friend. The murderess gave as a reason that she had killed her friend because she loved her and could not live without her; that they were engaged to be married, but that her wife to be had broken the engagement, etc. Her trial was probably the most remarkable one of the kind on record. She bore herself calmly throughout, manifesting no realization of her crime. When asked why she had killed her friend, she looked hurt and surprised, and cried: "Because I loved her, of course!" and burst into a flood of tears. She was found to be insane. On her way to the asylum she was taken to the grave of her friend, and displayed the extreme of grief. I have in my case-book the history of a young girl who conceived a violent affection for a nurse in a Pennsylvania so-called sanitarium. This nurse gave her massage, and thus they became acquainted. Their intercourse was by titillation of the vagina, or rubbing their naked bodies together. Curiously enough, on the same day on which the tragedy occurred in Memphis, Tennessee, other murders occured in St. Louis and Mobile, respectively, from the same perverted sexual passion. In the former city a Dr. C. T. Breedlove shot himself because his affection for Prof. Judson, one of the teachers of the High School, was not requited; whilst in the latter city a negress named Eleanor Richardson slashed her sweetheart, Emmie Wilkinson, another negress, because the course of true love had not run smooth. Adolphe Belot has lately written a curious novel upon this subject, entitled *Mademoiselle Girau—My Wife.*

Sexual hermaphroditism is a very rare condition, in which an individual at one period of life has the feelings and sexual desires of one sex and at another period of life has the sexual desires of the other sex. This may be conjoined with hermaphroditic defects or not.

By the term *masochismus* Krafft-Ebing designates a condition of sexual perversion in which a member of one sex takes great pleasure in being dominated by the other, so that the male has a sensuous feeling akin or almost akin to an orgasm in submitting to mortifying, humiliating, and degrading acts from the female. The name comes from that of the writer, Sacher-Masoch, who wrote a number of

novels upon this subject. The well-known French author, Baudelaire, was a sexual pervert of this class. Zola has depicted some masochistic scenes in his novels, *Nana* and *Eugène Rougon;* and many of the newer Russian novels deal with similar matters. Krafft-Ebing cites a number of remarkable cases of this kind, and shows how Jean Jacques Rousseau derived voluptuous pleasure in receiving blows from the hand of a certain Mademoiselle L.; and so pleasant were they to him that his ingenuity was continually exerted in provoking new punishment, and this when he was only a boy of eight years. He says that to be at the knees of a mistress, to obey her orders, to ask her pardon, was to him exceedingly sweet enjoyment. *Masochismus* in the female is either a very rare affection, as Krafft-Ebing knew only one case of it, or else the habitual domination of the female by the male would make it difficult to observe. And this difficulty would be enhanced by the innate reticence of the female about sexual matters, at least to male physicians.

Sadismus is the very opposite of *masochismus,* so that the member of one sex endeavors to give pain and exercise force upon the other sex. The term comes from the Marquis de Sade, whose obscene writings are well known to those acquainted with French literature.

Erotic fetichism is a form of sexual perversion in which sexual feelings are excited by a species of fetich being made of some article of wearing apparel, a lock of hair, etc., belonging to one of the opposite sex. Some obtain an orgasm by touching a piece of a garment, or a piece of silk or velvet, or stealing handkerchiefs, or shoes, or even by cutting off beads, or by throwing vitriol on women's dresses, etc.

Sexual metamorphosis consists of having the tastes and feelings and assuming the dress and habits of the opposite sex. Spitzka cites the instance of Lord Cornbury, a cousin of Queen Anne, son of Lord Clarendon, a member of the House of Lords, one time Governor of the colony of New York, who used to dress himself up in feminine attire and promenade upon the Bowling Green, as Spitzka graphically describes it, "with all the coquetry of a woman and the gestures of a courtesan."

The treatment of these cases of sexual perversion is very difficult. Hypnotism has seemed to be sometimes of value. Eastman has performed neurectomy of the pudic nerve in a female with masturbation and effected a cure. If the individuals afflicted are young, proper training and removal from vicious companionship will be sufficient; but perverted adults are difficult to deal with, unless some effective appeal can be made to their common sense, or unless their sexual perversion is conjoined with insanity.

CHAPTER V.

SIMULATION OF INSANITY.

Many words have been wasted upon the question as to how to detect simulation of insanity. The truth of the matter is that such

Fig. 165.

Stupid melancholia.

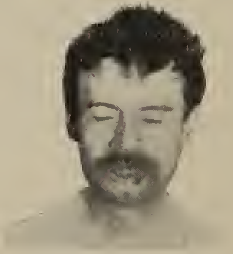
Fig. 166.

General paresis (maniacal stage).

Fig. 167.

Simple melancholia.

Fig. 168.



Melancholia attonita.

Fig. 169.

General paresis (terminal stage).

Fig. 170.

Terminal dementia.

simulation can only be detected by one who is acquainted, and thoroughly acquainted, with the different types of insanity, so that it can be

seen that the simulator is not giving an accurate picture of the form that he is attempting to portray. In many chronic cases of insanity, and in some of great intensity, the facial expression or *facies* is a matter of great moment. But this *facies* cannot be represented by photographs. Figs. 165–170 are portraits of different well-marked cases of insanity about which there has never been a question, and yet in some of them, especially in Figs. 165, 166, and 167, it would be difficult to detect that there was any mental aberration whatever.

CHAPTER VI.

THE PATHOLOGY OF INSANITY.

UNFORTUNATELY, at the present day our knowledge of the pathological alterations in insanity is in an embryonic state. Of certain of the insanities we have excellent information, whilst of others we have some glimmering of the facts, although of others still we must be said to know nothing at all. We know moderately well, for instance, the changes which take place in general paresis of the insane in alcoholic insanity, in senile dementia, and in some of the terminal dementias. We are beginning to know something of the facts in some cases of epileptic insanity, and in most cases of syphilitic insanity. Finally, we know nothing at all of the textural changes which are at the bottom of most forms of mania, melancholia, periodic insanity, paranoia, primary dementia, hallucinatory insanity, or the insanities of pubescence or of the puerperal condition. It will be my task to present in this chapter the general changes common to many of the chronic forms, and then describe those found in the various types in the chapters dealing with the latter.

The bones of the skull frequently present no abnormality, but when such are present they generally consist of thickening or increased density, or thinning. When they are thickened, this may be real or apparent, in the former instance being due to an increase of rarefied diploë, in the latter case from increased thickness throughout, as well as from subperiostitis. When there is increased density, this may occur with or without thickening, and the surfaces may be eburnated. In some exceptional cases the thinning may be so extreme as to render the bone almost transparent. In judging of these conditions of the skull, however, one needs an accurate anatomical knowledge, so as not to mistake normal conditions for abnormal ones. It is a curious fact that different portions of the skull will be found to share alike in these abnormal conditions; thus, the left frontal bone is often found to be thickened at the same time that the right occipital region is in the same condition. Very frequently there is found to be some hyperostosis. The subacute and inflammatory conditions are usually found in connection with increase in thickness of the cranial bones, and senile atrophy is frequently associated with decrease in thickness and density of the bones.

In chronic insanity the dura mater is frequently found to be adherent to the cranial bones, and, exceptionally, bony plates are found in its folds.

Thickening and opacities of the pia and arachnoid are frequent in the chronic insanities. Care must be taken, however, not to confound

this with the milkiness of the pia which is frequently found in the middle-aged and the aged.

The normal cerebro-spinal fluid varies between two drachms and two ounces; but in the chronic insane it may be increased to eight or ten ounces, buoying up the soft membranes and soaking them. This abnormal cerebro-spinal fluid is acid in reaction, whilst normally it is alkaline.

A frequent formation in insanities, especially of the chronic type, is the so-called arachnoid cyst. There has been considerable difference of opinion in regard to the exact genesis of these bodies. Calmeil, Boyle, and Virchow have regarded them as being due to an inflammation of the inner layer of the dura mater and to pachymeningitis interna. Bevan Lewis, on the other hand, maintains that they are of hemorrhagic origin, stating as his reasons that these cysts are readily removable, being but slightly adherent to the dura mater; that in the majority of cases there is no sign of pachymeningitis, as the dura is not thickened, softened, or vascular, and there is no organic connection between the cyst and the dura; that in the early stages they seem to be simple extravasations of blood into the arachnoid cavity; and, finally, that they coexist with recognized vasomotor disturbances, as the othæmatoma or insane ear. Bevan Lewis believes that they are due to an extravasation from the vessels of the pia mater.

The pia mater is frequently thickened, opaque, effused with lymph, and occasionally with pus, and frequently there is a meningo-cerebritis, so that the pia is adherent and tears away a portion of the underlying cerebrum when it is stripped off, leaving an eroded surface. In the more recent adhesions the cortex is apt to be pinkish in color, either generally or in limited areas, whilst the pia is thickened, its cells proliferated, its vessels tumid, and the overlying arachnoid also sharing in the molecular disturbance. Numerous spider like cells are found in the cortex immediately beneath the pia, in direct connection with its cerebral surface and with the vessels passing from it into the cerebral substance; and these spider-like cells are also found around the walls of the bloodvessels. When the adhesions are old there is a coarse fibrillar connection between the pia and the cortex, and this takes the place of the normally delicate neuroglia. That the spider-like cells are not found so frequently in advanced cases of insanity, such as senile atrophy, is supposed by Bevan Lewis to be due to the fact that they are comparatively early formations, having functions to perform, which, as we shall soon see, are not necessary in advanced cerebral disease; whilst in some cases they have succumbed to a fatty liquefaction.

The vascularity of the cerebral substance, both gray and white, varies very considerably in different forms of insanity, these variations probably being due not only to the particular form and its mode of onset, but to variable external factors. In some cases there is great congestion, in others none at all. In making the post-mortem it should be remembered that much will depend upon whether the chest is opened first or last, as in the former case the blood draining off from the thoracic vessels may very quickly make a great difference in the

vascularity of the cerebrum. The increased vascularity is sometimes limited to certain areas mapped out by the nutrient arteries, and these are usually found either at the junction of the white and gray matter or also at the region of the fourth layer of the pyramidal cells, this peculiar distribution being probably explained by what has been observed in injections of the cortex, viz, that the long, straight vessels and their horizontal end-works at the border of the gray matter are most readily filled, next the vascular end-work around the fourth and fifth layers, and lastly the vascular end-work in the third and the first layers respectively. These limited mottlings are found in the more acute forms of insanity or where the patient has died in an epileptic status. The mottlings are not necessarily pathological conditions, however, and in order that they should be so regarded there should be found with them minute extravasations of the small vessels, œdema of the tissues, and altered consistence of specific gravity, all of which should be evident to careful search with the naked eye, although, of course, microscopical examination may disclose disease which would not be otherwise visible. An anæmic condition of the cerebral substance is, however, most frequently found. Thus, uniform pallor cases prevailed in 841 out of 1565, or in 53.7 per cent. In melancholia, areas of pallor or diffused pallor are not infrequently found in the cortex supplied by the carotid system, whilst there may be hyperæmia in the area of distribution of the vertebral arteries. The most extreme forms of anæmia are found in chronic phthisis, or as the result of severe hemorrhage, as in the post-partum hemorrhage ushering in puerperal insanity.

Focal cerebritis, usually as the result of embolus or thrombosis, is commonly found in the insane, as is also chronic meningo-cerebritis, but diffused cerebritis is very rare. The tissue of cerebritis is usually not discolored, but inflammatory exudates will be found, and granule-cells, nuclei, leucocytes, and pigment, whilst the specific gravity will be increased. Close to such cerebritic patches there is frequently a white or yellow softening of a non-inflammatory nature, due to plugging of the minute vessels or to the pressure of the œdematous tissue. In most cases of insanity this cerebritis is due, as has been stated, to vascular changes. In general paralysis, however, the cerebritis spreads throughout the brain in areas that are not explained by any capillary distribution, in such a way as to indicate that the lesion is due to cellular change, more especially as the occipital convolutions are almost invariably spared, the changes being usually most marked in the frontal and parietal lobes.

Softening frequently occurs; thus, of 853 fatal cases of insanity examined by Bevan Lewis, 45.7 per cent. presented brains that were of normal consistence or above the usual degree of firmness, whilst 54.2 per cent. were softened, either as the result of disease or post-mortem change. Aside, however, from any putrefactive alteration, a lessened consistence of the brain is frequently found, although it must be noted that the cases examined have been mainly those of senile atrophy, general paralysis, and organic brain disease, such as block the large asylums. This lessened consistence is due to

œdema, structural alterations from senile fatty degeneration, widespread arterial disease, or inflammation. At all events, the vascular system is almost invariably involved in these cases. Before, however, it is determined that the brain is lessened in consistence, great consideration should be paid to the following factors: (1) the length of time after death at which the post-mortem has been made; (2) the condition of the temperature of the air; (3) the general condition of the subject. It is a very difficult matter to obtain firm brains in our large cities from the eleemosynary institutions filled with the half-starved and luckless outcasts of modern civilization, and it is especially difficult to obtain them in warm weather. Thus, the brain of the bomb-thrower who recently attempted to kill Russell Sage with dynamite was in a firm state fifty-three hours after death, whilst shortly afterward, in the month of April, when the temperature for two or three days had risen to 80° and 85° in the shade, I examined a brain from one of the institutions upon Blackwell's Island, of a man who had been apparently healthy until shortly before his death, and found it so soft as to make it useless for purposes of section. In another patient of mine considerable medico-legal interest hinged upon the question as to whether the brain was pathologically softened, as I thought it was, for the patient was a young man of thirty who died suddenly from cerebral syphilis in very cold weather, in the month of January, and yet only twenty-three hours after death the cerebrum was found to be so soft that neither hemisphere could be lifted by any large part of it without the dependent portions slipping away. When there are extreme degrees of softening, this can be readily enough determined, and one of the best tests of this condition is to see whether the brain-substance can be washed away by a gentle stream of water. The localized softenings which are found in insanity are very similar to those which are found in general softening of the brain (pages 306 and 307).

Atrophy of the cerebral gray and white matter frequently occurs after the acute insanities, and also in the chronic forms, and it may be either general or localized. It may be the result of an inflammatory process, from disease of the nutrient arteries, or it may be due to non-inflammatory cellular alterations, as in epilepsy.

Miliary sclerosis is a certain change in the cerebrum about which there has been great difference of opinion. Many very competent observers believe that these changes are artificial products, so-called *artefacts*, whilst others maintain that they are genuine pathological lesions, and among this latter number are Batty Tuke and Rutherford, who first described it in 1868; Kesteven, who wrote upon it in 1869; and Bevan Lewis, who has given an exceedingly minute and elaborate account of it in his recent work. Lewis states that this condition is found in perfectly fresh brains before any reagent has been applied. I can confirm Lewis in this latter observation, although I am not prepared to say that similar conditions may not be found in brains that have been preserved in alcohol. The best description is that of Bevan Lewis, just alluded to. Miliary sclerosis is usually

found in the white matter of the cerebrum, pons, and medulla, and the lateral columns of the spinal cord, and it is best studied in the latter site, although it is more often met with in the cerebral medulla. On holding up a stained section to the light, little bright pellucid points are seen scattered throughout it, just perceptible to the naked eye, and they are also seen by reflected light, but not by direct transmitted light. A low power shows that each of these consists of a lobulated patch, some twenty to fifty millimetres in diameter. In the brain they are limited to the white medullated structure, abruptly terminating as they approach the cortex, or, rarely, extending into the lower zone of the gray matter. The perivascular nuclei are often very greatly proliferated, many granular hæmatoidin masses cover the sheath of the vessel, and at a certain stage there is always an increase of the spider-cells. When present in the spinal cord miliary sclerosis becomes still more evident, and in many cases both lateral columns are filled with these minute bodies. The latter consist of colorless, translucent patches of molecular material, with a stroma of exceedingly delicate fibrils, and in them are frequently seen varicose medullated fibres, whilst they are surrounded by sclerosed tissue. All the medullated fibres in a line with the lesion are markedly diseased, end directly in the lesion, and are often regularly moniliform, the medullary matter being broken up into beads, or irregularly varicose, or it may lie in large pyriform masses, whilst beyond the fibres often lie naked and swollen axis-cylinders. Immediately surrounding these nerve-fibres are usually a large number of spider-cells. Tuke and Rutherford had regarded these changes as due to sclerosis, but the latter of these writers, after viewing Bevan Lewis's sections in 1889, agreed with him that these changes were due to a subacute inflammatory or degenerative change in the medullary nerve-tracts, at least in most cases.

There are genuine forms of sclerosis found in certain cases of insanity, however, besides the lesion which is designated as miliary sclerosis. Pozzi has described a form that he calls disseminated atrophic granular sclerosis, in which he found symmetrical lesions in the two hemispheres, particularly implicating the frontal and parietal lobes and the paracentral lobule, and isolatedly in other parts of the brain. The convolutions were atrophied and as if wormeaten and granular.

So-called *colloid degeneration* of the brain consists of certain round or oval bodies from six to twelve or even forty millimetres in diameter, often found in the brain and spinal cord of the insane in great numbers. Like miliary sclerosis, there is a great difference of opinion as to whether these are artificial productions or genuine lesions. They are homogeneous, devoid of concentric markings, colorless and pellucid, slightly tinged by hæmatoxylin, but unaffected by carmine or aniline dyes, and exhibiting no reaction to iodine or sulphuric acid. They are seldom found in the gray matter, and when they are, it is only in the large medullated tracts, or, more rarely, in the intracortical arciform fibres. They follow the course of the medullated fibre, and the axis-cylinder can often be traced through

the centre of the swelling, or in other cases a pyriform body is penetrated by a swollen axis-cylinder, or, again, the medullated fibre may seem to terminate within the swelling. These bodies often have free nuclei attached to them, which seemingly come from the spider-cell. This colloid body is attached to the medullated fibre, and is not removable like the miliary lesion, and under certain conditions they become opalescent or granular, swell up, burst their albuminous sheaths, and swim around in masses. Bevan Lewis gives the same explanation of their origin as that which he offers for miliary sclerosis.

In insanity there is frequently a granular disintegration of nerve-cells, so that they become swollen and more spherical, lose their homogeneity, and show granular formation, whilst the protoplasm and nucleus tint but faintly with carmine or aniline dyes. The nucleus often shares in the degeneration, becoming angular or distorted, whilst the ordinary pigment of the cell, which is usually collected at its base, becomes diffused, and the cell takes on a yellow hue. The lateral processes and the apex, too, are affected, and dwindle and disappear; but, curiously enough, the process from the base generally remains, although it may be swollen and without a miliary sheath. In many cases, too, the cells are totally disintegrated, their site being marked by a granular mass. The bloodvessels also partake of the change. The perivascular spaces become enlarged with fatty granules and deposits of hæmatin, and the vessels are generally atheromatous and covered with fatty granules. The cerebral parenchyma around the cells and the vessels is, as a rule, full of fatty granules. The first description of this granular degeneration was given by Major. It is especially frequent in senile atrophy.

Pigmentary or fuscous degeneration of the nerve-cells is frequently found in epileptic insanity and general paresis. In order to estimate this, however, an acquaintance is needed with the usual amount of pigmentation of the nerve-cells, the gray matter of the brain and spinal cord. A fresh cell of the cortex, examined in a section prepared by the microtome, presents at one of the inferior angles or along the base a small collection of golden-yellow pigment, from which a number of dark amorphous granules are scattered; and, although this pigment is surrounded by a protoplasm, it is yet quite distinct from the latter, and it often encircles the nucleus concentrically. In the pathologically pigmented cell, however, the whole cell becomes tumid, and instead of being elliptic, is of pyriform or spheroidal contour. The pigment is greatly increased in quantity. The cell protoplasm stains intensely with aniline blue-black, and, if subjected to this dye for more than a very short period, the unpigmented protoplasm and the nucleus become obscured. At the same time the pigmented portion is entirely unaffected by aniline or carmine dyes, and becomes of a bright yellow or brownish-yellow, and has a rough, granular look. The nucleus is deeply stained by the usual reagents. As the pigmentary degeneration proceeds, other changes ensue. The nucleus, probably from retraction of the unaffected protoplasm, is drawn toward

the apex, or laterally, or even toward the base. This nucleus may be pigmented, but it usually loses the oval contour and becomes more or less irregularly angular. The staining by aniline may grow to be less evident. The lateral process may dwindle or disappear. The pigmented area appears to be separated from the rest of the cell by a capsule of more deeply stained material, and at this period there may be one or more highly refractile spots in the nucleus. Finally, there is a general shrinking of the cell, or the cell may be filled by a somewhat bright, translucent, colorless material, its outline may be so faint as to cause it to be easily overlooked, and vacuoles may appear. The granular pigment in fresh sections is unaffected by ether, alcohol, caustic soda, or fuming nitric acid. When the cells are well advanced in degeneration, this pigment may be decidedly darkened with a one per cent. solution of osmic acid, and a small portion of fatty matter may thus be evidenced.

Bevan Lewis has called attention to an arrest in development of the nerve-cells in certain cases of epileptic idiocy and imbecility, especially in the second and third layers of the cortex. These degenerated cells are normal so far as regards contour and branching, but degenerated by the granular, pigmentary, or fatty conditions of their contents, and they are completely unaffected by the staining reagent employed. That it is not a retrogressive process is maintained by Bevan Lewis, because he thinks that if the granular, pigmentary, or fatty condition were a diseased process, this would be progressive and result in complete disintegration, and also because the nucleus of these cells is usually displaced, so that instead of being at the junction of the apex process of the cell, it is often pressed and flattened against the sides.

The formation of vacuoles in nerve-cells is always associated with granular degeneration, although granular degeneration may exist without vacuolation. Vacuolated nerve-cells are met with in senile atrophy and in chronic alcoholism. The cell contains oval or spheroid bodies, colorless and lustrous, highly refractile, unaffected by any staining reagent, and in some cases, when the refractile quality is wanting, the contents of the vacuole will be seen to have escaped by rupture. It has been suggested that this condition of vacuolation is due to defective oxidation, inasmuch as it occurs in phosphorus-poisoning, which induces a fatty degeneration from increased metamorphosis of albumin, but chiefly from interference with the oxidation of the tissues through destruction of the red blood-corpuscles. The experiments of Voit, Bauer, Popow, Danilo, Kreyssig, and Flesch upon dogs and rabbits also indicate that phosphorus and arsenic produce a granular degeneration and vacuolation of the ganglion-cells of the spinal cord. Flesch and Trzebinski attribute the change to the reaction of chrome-preparations, and the latter states that the lesion was never observed by him in fresh preparations, although Lewis flatly contradicts them both. In some cases the nucleus alone is vacuolated, which occasionally occurs in the small, angular cells of the second layer of the cortex, and in one case sketched by Bevan Lewis the change was so extensive that almost every cell was affected.

Whether it is important or not to make a distinction between vacuolation of the cell and vacuolation of the nucleus is at present undetermined, but Bevan Lewis thinks that there is a difference in the pathogenesis of each lesion, because he regards it as improbable that defective oxidation should affect only one layer of nerve-elements, and this the smallest, as it seemingly does in vacuolation of the whole cell, so that here presumably the cause is in some textural change in the cell itself or its connections.

Some extremely valuable and interesting observations upon the blood of the insane have been made by Macphail, and to this essay the prize and the bronze medal of the Medico-Psychological Association of Great Britain were justly awarded in 1884. Examining a number of cases of general paralysis, epilepsy, dementia, and many unclassified cases of insanity, he arrives at the conclusions that while there is no evidence that anæmia in itself is the cause of insanity, yet an anæmic condition of the blood is undoubtedly in many cases intimately associated with mental disease; that the blood in the demented class of asylum patients is deficient in hæmoglobin and in hæmocytes, and that deterioration progresses as age advances; that the blood of patients known to be addicted to masturbation is deteriorated in a marked degree; that the blood is below the normal standard in general paralysis, and the deficiency is greater in the active and completely paralyzed stages of the disease than in the intervening periods of inactivity and quiescence; while there is a deficiency in the quality of the blood of epileptics, the decrease is not so pronounced as in ordinary dements at the same age; that prolonged and continued doses of bromide of potassium do not cause deterioration in the quality of the blood; that prolonged attacks of excitement have a deteriorating influence on the quality of the blood; and that the blood of the average number of patients on admission is considerably below the normal standard. He also found that in those who recovered the quality of the blood was improved during residence in the asylum, and that on discharge it was not much below the normal standard. There was also a close connection between gain in weight, improvement in the quality of the blood, and mental recovery; and while there was a definite improvement in the condition of the blood during mental convalescence in all cases, the improvement was both more pronounced and more rapid in those who had had tonic treatment. He found four tonics to be most efficacious, either alone or in combination, in restoring the quality of the blood, and these were in order of their value: iron and quinine, iron alone, strychnine, and malt-extract. Arsenic was of little value as a blood tonic in these cases, and quassia and cod-liver oil did not give satisfactory results.

CHAPTER VII.

MELANCHOLIA.

Definition. Melancholia is a form of insanity in which there is a profound melancholy, the cerebral reflexes are lessened and attended with great mental distress, and there is often a suicidal impulse.

Clinical History. Melancholia may be divided into four forms:

1. Simple melancholia;
2. Agitated melancholia, or melancholia agitata;
3. Hallucinatory melancholia;
4. Melancholia with stupor.

The simple form may, in its slighter degrees, bear an almost indistinguishable resemblance to an ordinary depression of spirits, and yet, even in the slighter cases, there is a peculiar visage of mingled woe, shadowy mental dulness, and distrust, that is instantly detected by an experienced eye. Relatives and friends, so intimately acquainted with the patient as to be competent to notice mental shades of difference, will observe something wrong, although they cannot always define it. Even in these slighter degrees the mental reflexes are attended with mental pain; in other words, any impression coming to the cortex from without is painful. The sights, sounds, and sensations that were wont to give pleasure now cause pain. Wife, children, other relatives, friends, pleasant sights, harmonious sounds, agreeable odors, savory savors—all produce mental distress. The most agreeable or mirthful object cannot reach the mind without metamorphosis, in its passage through the melancholiac cortex, into something mournful. It is almost impossible, therefore, to make the patient smile. There are certain symptoms of great diagnostic value in this simple melancholia, as I have discovered, viz.: a peculiarity of the melancholia, an obstinate insomnia, and peculiar sensations (to which I have given the name of the *post-cervical ache*) in the back of the head and neck, or at the vertex, or up and down the spine. With these I shall deal more in detail a little further on. In this simple melancholia the memory is usually maintained, although at the acme of the disease in some cases there may be a slight impairment, so that the events of this period are afterward remembered mistily. The reasoning faculties, so far as the melancholia permits, are usually intact, so that the patient can reason well enough except as the painful mental reflexes warp the mind by converting all sensations into painful ones. Simple melancholia is usually without hallucination or illusion, except in the most marked types, and even then the hallucinations or illusions last only a short time.

In hallucinatory melancholia the melancholy is much more pronounced. The patient has, in addition to the painful mental reflexes, distressing hallucinations and illusions. He lives in a realm of terror. Not only does the diseased cortex make painful the myriad ordinary sensations travelling in from the various sensory structures of the body—retina, ear, tongue, nostrils, skin, mucous membrane, genitalia, viscera—but it converts them into hallucinations and illusions of the most terrifying character, and according to the degree of the melancholiac cortical transforming power is the amount of mental distress manifested by the patient. Some break out with occasional exclamations of suppressed agony, as, "My God, I cannot stand this any longer." Others will tell you, with tears streaming down their

Fig. 171.

Melancholia agitata.

cheeks, that they have committed the unpardonable sin, and that they are damned in the eyes of God and man. Some will have paroxysms of great distress. Others will be in constant agitation, wringing their hands, praying, beseeching, tears streaming down their cheeks, incoherent, perhaps violent (*melancholia agitata*). (Fig. 171.)

Melancholia with stupor is a form in which the stupidity comes early in the disease, and it is to be distinguished by this feature, as well as by the degree of the melancholia, from the slight temporary stupidity which sometimes supervenes in convalescence from the other forms of insanity. In this form the patient sits mute, motionless, almost expressionless (*melancholia attonita; angedonnerte Melancholie* of the Germans). (Fig. 172.) Occasionally there are slight cataleptoid symptoms. The silence may at times be broken by some muttered expressions of mental distress. When the patient

is addressed, it is evident that he is confused mentally, and he will look at one with a bewildered expression. Little as the face indi-

FIG. 172.

Melancholia attonita.

cates it, however, these cases are tormented by the most frightful mental terrors, and the patients will afterward speak of this period with horror when they have retained memory of it. Fortunately,

however, in this melancholia with stupidity, as well as in hallucinatory melancholia, the memory is generally impaired throughout the worst periods of the disease.

In any of these forms the patient may have certain vague distressing sensations in the præcordia, causing great fear, and sometimes productive of sudden outbreaks of violence. (This is the so-called *præcordial fear* of the Germans.) These sensations are probably ordinary præcordial sensations, as of palpitation from intestinal reflex or functional cardiac trouble; and when we consider how apt they are to cause alarm in the perfectly sane, we can imagine with what terror they will inspire a melancholiac as they are transmitted to his woe-producing cortex. All cases subject to this præcordial fear are dangerous.

All these forms of melancholia have certain peculiarities in common, namely:

Suicidal tendencies;
Violent outbreaks;
Self-limiting or developmental tendencies;
Occasionally præcordial fear.

The slighter the melancholia, the less will be the mental distress, and, therefore, the less will be the suicidal tendency; but even in the slightest cases these patients cannot be trusted, because a suicidal impulse may develop suddenly, and it is precisely in these milder cases that the most deliberate and carefully planned suicides occur. A patient of this mild type, whom I saw many years ago in consultation, was regarded by his family as entirely harmless, yet one day when his daughter left him for a moment to go into an adjoining room, he slipped out, walked over two miles to the park, around which he tramped for fully an hour, until he had selected, with excellent judgment, about the most secluded spot in the grounds, then tore up his shirt, made a rope of it, and hung himself to a branch of a tree. Another patient, although occasionally agitated, was thought to be so harmless that former precautionary measures were abandoned. She had consented quite cheerfully to go to an asylum, as the family means were limited, and had made her arrangements quietly and docilely. Just before starting her mother attempted to lecture her, in a mild, motherly way, upon the necessity of being more cheerful, when she became greatly excited, suddenly drew out a wisp of paper from her axilla, took the end of a match from under her long finger-nail, where she had secreted it, lit the paper, and set fire to her nightgown, sustaining a frightful burn on the thigh and abdomen. Under proper treatment, however, this suicidal impulse soon disappears from the milder forms.

Violent outbreaks of a homicidal nature are usually only observed in the graver forms, as in the agitated melancholia and the melancholia with stupidity.

The milder forms of melancholia can be distinguished from neurasthenia by means of the three symptoms to which I have called attention, namely, the peculiar *facies*, the insomnia, and the post-cervical ache. The *facies* is something that cannot be represented

by a photograph, as I have already said, because the very act of consciously looking at an object, as in having a photograph taken, disturbs the slight unconscious play of the muscles that makes the expression. In very marked cases of this form, however, the *facies* will be so pronounced that it is apparent to anyone, as in a recent well-known case that figured in the courts, in which the question of shamming was brought up, and concerning which I was asked whether it was not possible for an actor to assume the patient's expression; to which my answer was that while an actor might assume it for a short period of time, it would be beyond human powers of imitation to continue it for days and weeks, much less months. This peculiar *facies* is made up of an expression of gloom and suspicion. The post-cervical ache is the name which I have given to a peculiar complex of sensations that these milder patients usually experience, generally in the occiput and in the upper part of the neck, although in exceptional cases the sensation may extend up to the vertex and even over to the brow, or in the other direction down the whole length of the spinal cord. The sensation varies so from patient to patient that it is impossible to give it an accurate name, as one will call it an ache, another a pain, or a thrilling sensation, or a frightened feeling, or a creeping, or a throbbing, or an uneasiness, and so on through all the gamut of sensory perceptions. In 175 cases of this disease which I have examined all three symptoms were present in 54 per cent., whilst in 18 per cent. the post-cervical ache was lacking and the insomnia was absent in only ten cases. The insomnia varies greatly in degree. In some cases it may be so great that the patient will only obtain an hour or two of sleep for months, while in others it may merely cut off half an hour of slumber, or even occur from time to time, or for a short period at the onset. Usually it persists for months after the other symptoms have disappeared, and if not properly treated, it may last for years; indeed, I attach so much importance to this symptom that I never consider a patient cured until the tendency has been thoroughly eradicated. In some few happy cases, however, it disappears with promptness, and does not return. It has been objected to my discovery of these three symptoms that they are not always present, and that even if they are, one or more of them are often found in other mental diseases. To the first objection I would answer that I have yet to see the first case of indubitable simple melancholia in which two of these three symptoms have not been present, and I think that my opportunities of seeing such cases are sufficient to warrant me in attaching a value to this fact. In reply to the second objection, my critics do not seem to understand the very simple fact that I do not claim for these three symptoms *separately* the diagnostic value which I do claim for them when grouped together. We should never forget that just as we have so many letters in our alphabet, from which in varying combination are formed the different words that constitute our language, so have we so many symptoms in medicine which can be readily catalogued, and from the varying combination of which we make our diagnoses of the different diseases; and that just as the individual letter is of small

importance, so is the individual symptom of relative insignificance. In addition, it should not be forgotten that melancholia, the disease, is not melancholy, the mood.

These milder forms of melancholia are self-limited, lasting from three months in the very mild cases that are properly treated, to nine and twelve months or even longer in the severer cases, especially in those occurring in elderly and broken-down individuals. All these forms of melancholia have a slight developmental tendency, so that it is not always possible to prognosticate the gravity of the disease until it has been established for several weeks.

CAUSATION. The causes of melancholia are—

- Heredity ;
- The emotions ;
- Mental and physical overstrain ;
- Surgical operations ;
- Inanition ;
- Pelvic disease ;
- Intestinal derangement ;
- Febrile diseases ;
- Parturition ;
- Derangement of abdominal viscera ;
- Diabetes.

Although melancholia is sometimes of hereditary origin, it occurs much more frequently without heredity ; and even in the cases where there is heredity this may consist of melancholia itself, a predisposition to insanity, or a neurotic tendency.

The emotions are, if I may judge by my own experience, by far the most frequent of all the causes of melancholia. This is contrary to the general belief, which is that one of the diagnostic signs of melancholia is the causelessness of the melancholy ; but I can confute this with many cases from my case-book. One of the most typical cases that I ever saw was caused in a mother by the death of her only child, whilst another followed directly upon the death of a friend, another because of jealousy of her husband, and so on.

Mental and physical overstrain is a frequent cause, and this fact also contradicts the belief in the causelessness of melancholia. Indeed, I think that the two most frequent causes of this mental disease are mental and physical overstrain and the emotions.

Surgical operations occasionally cause melancholia. I have not, however, thought it proper to classify the cases of melancholia following operations in a separate chapter, for they differ in no wise from melancholia from other causes. It is a curious fact, too, as I shall have occasion to point out, that surgical operations will sometimes relieve melancholia.

Inanition is a rare cause.

Pelvic disease in the female is frequently causative of melancholia. It is a great mistake, however, for gynecologists to assume, as they so often do, that relief of pelvic lesions will of itself cure the melancholia. This will sometimes happen, but it is much more frequently the case that the melancholia must itself be treated, and it will often

disappear in spite of the persistence of the pelvic lesion which has been causative of it.

Intestinal derangement often causes melancholia; and the most frequent of these intestinal lesions is a functional derangement of the duodenal region or of the small intestine. It is true of these causes, as it is of pelvic lesion, that relief of them will generally not be sufficient to disperse the melancholia. I made many failures in my earlier years because of my belief that this type of melancholia could be directly relieved by cure of the intestinal derangement, but a wider experience has taught me to be very cautious in this matter.

The febrile diseases will sometime cause melancholia, too, but more generally the post-febrile insanity is a mild form of hallucinatory insanity or paranoia.

Parturition not infrequently causes melancholia. It is absurd to class the insanities following parturition under the head of puerperal insanities, for they differ in no wise from the same types of insanity from other causes.

Melancholia sometimes follows nephritis or diabetes, but rarely.

These different causes, however, are far more prone to cause the simple form than they are to induce melancholia agitata and stupid melancholia.

Diagnosis. The diagnosis is usually easy except at the outset of the mild cases, or at the period of a mild onset of the severe cases; and it is just at this time, especially in simple melancholia, that the three symptoms of the *facies*, insomnia, and post-cervical ache are of enormous value, together with the fact that in certain cases there is a slight dulness of the cerebral reflex. It is sometimes stated that the fact of the melancholia having been causeless is a diagnostic symptom; but this is a great mistake, because, as I have already stated, melancholia, of all the mental diseases, is most prone to develop after a great emotional shock, such as the loss of some one very dear to the patient, or business reverses, or some great cause for worry and anxiety. But even in these cases the sadness is usually greater—is, so to speak, more morbid—than is warranted by the cause. In the severer cases the hallucinations, delusions, stupor, cataleptoid symptoms, agitations, præcordial fear, and the violent outbreaks will make the diagnosis very certain.

Melancholia is to be differentiated from—

- Sadness due to grief or anxiety;
- The initial depression of mania;
- Hypochondria;
- Hysteria;
- Katatonia;
- Insanity of doubt;
- Periodical insanities;
- Primary or secondary dementia;
- Endarteritic insanity of middle life;
- General paresis;
- Grave brain-disease, such as tumors, intracranial syphilis, disseminated sclerosis, and meningitis.

Sadness from grief or anxiety seldom makes a patient so mechanical and lethargic as does true melancholia. Great shock of a mental nature may stun a patient for a certain period, but it will be usually succeeded by a certain outbreak of tears or manifestation of grief, and about such a person there is not the quiet unreasoning melancholy of a melancholiac, nor is there apt to be marked insomnia, and never any post-cervical ache or the dull cerebral reflex.

The initial impression of a mania is usually not a true melancholy, but is of the nature of depression of spirits, such as we constantly see at the outbreak of some sickness, and it differs entirely from the *facies* and behavior of a melancholiac. Besides, the supervention of mania, usually in a few days, will make the matter plain.

A hypochondriac seeks relief, constantly talks about himself and his woes, and rushes from physician to physician; but the melancholiac is reserved, quiet, asks for no treatment, and is frequently very much averse to it.

Hysteria may simulate melancholia quite closely for the time being; but the caprice in the symptoms, the other signs of hysteria, and the overdoing, so to speak, of the simulation, will make the diagnosis easy to any good observer who has ever observed a melancholiac.

Katatonia can be readily differentiated by its commingled symptoms of marked catalepsy and great stupidity.

The insanity of doubt may sometimes have conjoined with it slight melancholic tendencies, but even then the constant repetition of certain vague fears that nothing can satisfy, together with the absence of the *facies*, insomnia, and post-cervical ache, will make the diagnosis plain.

A periodical insanity can, of course, be only diagnosed when it has returned once or twice, but the circular form, in which melancholia immediately precedes mania, can be suspected from this very fact; moreover, the melancholia is not accompanied by the three symptoms jnst spoken of, if I may judge by my experience in a few cases.

Primary and secondary dementia can be readily recognized by the silliness of the patient, either coming primarily or being secondary to some brain disease. Insanity occurring in people of middle age, usually associated with chronic endarteritis, cardiac hypertrophy, and chronic interstitial nephritis, is very seldom a true melancholia, but usually consists of depression of spirits alternating with more or less capricious and confused acts, outbreaks of temper, and eccentric conduct. (*Vide* chapter on "Dementia.")

General paresis of the insane, or paralytic dementia, is to be distinguished by the unequal pupils, the lingual, facial, and bodily tremor, the extravagant delusions, the imperfect speech, and finally the paretic symptoms.

Any gross brain disease may of course produce depression of spirits, but it would be inexcusable to confound this with a true melancholia, and the symptoms of these gross brain diseases will be found in their proper chapters under the head of "Tumors," "Intracranial Syphilis," "Multiple Sclerosis," and "Meningitis."

Prognosis. Prognosis is good in simple melancholia and in the milder forms of hallucinatory melancholia, except in the aged, when it is usually exceedingly intractable. It is doubtful in melancholia with stupidity. Melancholia agitata occurring in slight degree with simple melancholia is of good prognosis, although the disease will be prolonged by it. When it occurs alone, however, and is severe from the start, it is of doubtful prognosis. The duration of the disease varies from a few weeks to two or three years, the average being about nine months in the curable cases.

Treatment. All these cases of melancholia should be isolated, either in their own house or in an asylum. It is a great mistake to have them travel or force them to seek amusement, as is so often done in the early stages; for these efforts at amusement are extremely painful to the patient at this excitable though melancholy period, whilst they may be beneficial in the later stages of convalescence, when the painful mental reflex has become much more healthy. The simple form of melancholia may be readily treated at home, in a boarding-house, or a hotel, if proper watch can be kept upon the patient, either by some member of the family or by a nurse. This is also true of the milder forms of hallucinatory melancholia, but the severer cases of this form and most cases of melancholia with stupidity are best treated in an asylum, unless the means of the patients are so ample as to enable them to employ one or two nurses for a year or more.

Opium is the best of all drugs for the direct treatment of the melancholia. I use only the aqueous extract of opium, made up into carefully triturated tablets. One-eighth of a grain is taken once, twice, or even three times a day, according to the severity of the melancholia and the susceptibility of the patient. Great care must be taken to have the opium pure. It must never be bought haphazard in the shops, but should be obtained from some reliable source and then analyzed. Morphine will sometimes answer the purpose as well, although frequently it excites the patient and constipates, which latter symptom is comparatively infrequent with the opium. To overcome the restlessness that is often so prominent a feature, the bromides should be employed, preferably of potash or sodium, usually giving from twenty to thirty grains at bedtime in the milder cases, or oftener in the severer ones.

The great plague of the melancholiac is insomnia, and to overcome this the following drugs may be used, the choice being indicated by the order in which they are named: Sulphonal, chloralamid, urethan, paraldehyde, chloral hydrate. Of sulphonal the dose should be thirty grains, repeating it in an hour if necessary. It should be finely powdered, as it is very insoluble at the best, and given in a cup of chocolate, milk, or beef-tea an hour before bedtime. But it is best in the compressed tablets. The dose of chloralamid should be 10 to 30 grains, in the form of compressed tablet or an elixir. Urethan is very soluble, and can be given in 10- to 20-grain doses. Paraldehyde is best administered in a mixture, the dose being 1 to 2 drachms. Chloral hydrate is probably the best of all these hyp-

notics, but is very apt to be depressing, and melancholiacs have a strong tendency to form the chloral-habit. The initial dose should never be beyond 10 grains, and it should only be repeated cautiously.

In the form of melancholia which is caused by intestinal derangement the latter should always be carefully treated. The best treatment is usually the dilute muriatic acid, 20 drops three times a day in a wineglass of water after meals, together with an occasional dose of calomel, 3 to 5 grains at bedtime, to be followed in the morning by a laxative, such as two teaspoonfuls of the sulphate of magnesia, or a glass of Hunyadi or Rubinat. When this treatment has been continued for about a week, it will generally be found advantageous to give 2 grains of salol three times daily, one hour after meals, in the form of tablet triturate, capsule, or a pill, together with 3 grains of a reliable preparation of pancreatin. Constipation should be carefully overcome in this form by means of the aloetics and the laxatives which have been spoken of in Chapter IX., under "Neurasthenia."

If the general health of the patient is depressed, it should be carefully remedied by attention to the measures that have been advocated under "Neurasthenia;" although rest is usually not at all applicable in a case of melancholia, as the melancholiac is usually too restless to submit to it. In all cases, however, rigid isolation of the patient should be maintained, for it must be remembered that these melancholiacs have, combined with their melancholia, a high degree of nervous irritability, so that I have frequently seen relapses brought on by a disregard of this precaution. No attempt should ever be made to amuse a melancholiac, for every well-meaning endeavor of this kind simply increases the nervous erethism and fails entirely of its effect. Wherever it is possible to do so, I put patients of this class in charge of a trained nurse, because they are more easily controlled by strangers than they are by their own family; besides which, it is almost impossible to make the family realize that a person who can converse intelligently and whose memory is so little impaired should not be made the recipient of all the little carking cares that have no effect upon a well person but that simply aggravate the melancholia. When a nurse is in charge of a patient the family should be excluded entirely. Indeed, in many cases of pronounced melancholia in which the family relations are sources of worry and anxiety, I positively refuse to treat the patient unless this isolation can be obtained.

These cases of melancholia, especially of the frequent simple type, are very prone to relapse, and they should be carefully kept under treatment until this tendency has been obliterated. Even after this there will be a marked disposition to insomnia, and the peculiar sensation to which I have given the name of the post-cervical ache will continue for years. The tendency to insomnia is always a source of anxiety to me, but the continuance of the post-cervical ache is a matter which I regard very lightly, and I always instruct my patients when they leave me to return promptly if the obstinate insomnia should again supervene. The post-cervical ache is often a

very distressing symptom, inasmuch as the sensations, which are generally slight, may become actual pain at times. For an exacerbation of this post-cervical ache the best remedy is that to which my attention has been called by Dr. Bauduy, of St. Louis, namely, phenacetine, 5 to 10 grains of which will often act like a charm. I am sorry to say, however, that the effect of this is only temporary. A much more permanent effect upon the ordinary less sensations can be obtained by galvanization of the brain. The current should be applied every day or every second day in the manner that has been described upon page 123, a current of 2 to 3 milliampères being used, this being continued at first for three minutes and afterward for five.

I desire also to impress upon my reader the necessity of not permitting the patient to know the nature of the medicines that are given. It will be very difficult usually to prevent him from recognizing the bromide of potash, because most intelligent people are familiar with its taste. But I never give a prescription to the patient for the opium and morphine or the hypnotics. I send these prescriptions myself to the pharmacist, mark them, "no copy or repetition," and I never deal with a pharmacist who will not respect this injunction. In the many cases, however, that I have treated I have never yet seen the habit formed of taking these drugs. At first I thought that this was due to my precautions, but I have since come to believe that there is a curious law at work in these indivduals which prevents the person who really needs hypnotics from forming the habit for them, just as the late Dr. Austin Flint used to say that he had never seen an inebriate made in his series of several hundred cases by his constant habit of ordering large doses of alcoholic stimulants in tuberculosis. Nevertheless, I would again strenuously advise the physician to take the precautions which I have done. I do not believe that a single one of the hundreds of patients whom I have treated has the faintest idea of what medicines have been given him. They are frequently very inquisitive, but I tell them bluntly that, as they can know nothing of the action of drugs, it is not proper that they should be informed, and I never have any further trouble

Melancholiac females are almost always worse at the time of the menstrual period, and its approach can generally be detected by the increased restlessness, deepened melancholy, and disturbed sleep of the patient. In all cases, therefore, the patient should be kept in bed as much as possible until the menstrual flow has been well established. She should be watched very carefully at this time, and in some cases the dose of medicine should be temporarily increased, diminishing it again in a few days.

Surgical operations upon the pelvic organs of the female, especially in cases where the melancholia has been seemingly caused by lesion of these, will often have a remarkably beneficial effect if done at the proper stage of the disease. The patient should have entered upon the stage of convalescence and the withdrawal of the medicines should have begun. In some cases, too, a tardy convalescence will be

affected in a marvellous way for the better by an operation upon the female pelvis even when there is no lesion of it; so that I often have my gynecological friends operate upon some slight abrasion of the os, curette the uterus, or do some minor operation in these cases. This last observation, however, is more applicable to cases of insanity and hallucinatory mania than to those of melancholia.

The question of committal of a case of melancholia is often one that involves a great responsibility. My rule is never to commit a patient who can be treated at home, for I am no believer in the asylum treatment of curable cases of insanity. In melancholia, as well as in many other forms of mental disease, the perceptions and the memory are frequently unimpaired or but slightly affected, and the patient either has a full realization of the fact that he or she is in an asylum, or they will in the course of time awaken to such a realization. The nurses of a lunatic asylum are never so efficient as the trained nurses whom we are now fortunate in possessing in such numbers, nor are they of as high a class in point of refinement and education. Even if they were, they are under no such stimulus or oversight as when they are watched by the keen and anxious eyes of an affectionate mother, daughter, relatives, or by an attending physician. Besides, the convalescence of patients in a lunatic asylum simply brings them face to face with what is generally regarded—whether justly or not—as one of the horrors of life, namely, a lunatic asylum. Then, too, however slight their mental affection may have been, the fact that they have been committed to a lunatic asylum clings to them through life. Like Lady Macbeth's "damned spot," that would not out, it too frequently blasts their career, whilst a severe attack of mental disease treated at home is lightly regarded by the laity under some such soothing euphuism as brain fever. Nor do I believe that the stage of convalescence can fail to be retarded by the constant contact with lunatics. For all these reasons I never commit a patient to an asylum, as I have said, if he or she can be treated at home. The question of whether the latter can be done will be determined by the degree of insanity, the probable duration of it, the environment of the patient, and his or her circumstances.

CHAPTER VIII.

MANIA.

Definition. Mania is a form of insanity in which the cerebral reflexes are increased and attended with great mental pleasure.

Clinical History. The prodromata of mania are vague and indistinct, and consist of symptoms pointing to reflex cerebral irritation, such as restlessness, vague forebodings, eccentric conduct or words, flushing or pallor of the face, disturbed sleep, and depression (not melancholia). It is not always easy to distinguish these prodromal symptoms from moodiness or depression due to many ailments, though generally something that is done or said will be so at variance with the patient's character as to excite the suspicion of some watchful female, wife, sweetheart, or mother, or even of some intimate male friend or business associate. The impressions of such witnesses, even though they cannot always bear radical analysis, should never be disregarded.

The forms of mania are—

Simple mania;
Hallucinatory or delusional mania.

In the simple form of mania the patient talks, acts, and feels like a person who is slightly under the influence of some stimulant. The mental operations are quickened, and they are pleasurable. The mental condition is the very antithesis of that of melancholia, for whilst it is impossible in the latter to excite a pleasurable emotion, it is almost impossible to make a maniac melancholy. To such a patient the world seems roseate-hued. He laughs, he jests, he is possessed of an infinite good humor, at the same time that his remarks and repartee are smarter and clever than is his wont. His physical activity is also increased, and he is restless. Careful observation will, however, make it very evident that this increased mental and physical activity is unreal. A slight mental confusion may be detected. The eye may have a dull and heavy expression, or there may be a weary look, the face being oddly at variance with the pungent promptness of the conversation. In this simple mania delusions, illusions, and hallucinations are usually entirely absent, or slight; in the latter event the patient has seemingly some vague, dimly perceived delusion or hallucination, whose unreality he himself recognizes, or out of which he can be readily reasoned for the time being.

In hallucinatory or delusional mania the hallucinations and delusions are not only much more marked, but the increase of mental and physical activity is apt to be greater, and the patient becomes confused and incoherent. In the cases running a subacute course from the beginning, however, the incoherence and mental confusion may

almost or entirely disappear, whilst the hallucinations and delusions persist.

Mania may be acute, subacute, or chronic. The acute form is generally one of great excitement. The patient talks incessantly, and is in incessant motion. He is seemingly violent, and may be provoked by a trifle to the most outrageous act. His cerebral reflexes are enormously exaggerated, but he is not inherently violent, and can be easily controlled by an expert attendant, differing in this respect from the patient with the acute delirium of alcoholism, of gross brain disease, or of delirium grave. The subacute type is that usually assumed by simple mania. The chronic form is usually preceded by the acute, rarely by the subacute.

The tendency of mania to develop is slight. Patients generally show the worst of their complaint in the first week or two.

Duration. The duration of simple mania under proper treatment will be from three to six months, but hallucinatory mania usually lasts about eighteen months, and sometimes for years.

Causation. The causes of mania are very much the same as those of melancholia, although the emotions, the intestinal derangements, and diseases of the female pelvis do not play so prominent a part. Mental and physical overstrain, febrile diseases, parturition, surgical operations, and inanition are, however, of about the same relative importance.

Prognosis. The prognosis is good in simple mania. In all the different forms the favorable factors are: subacuteness, moderate increase of cerebral reflexes, slightly defined delusions and hallucinations, and a reasonable proportion between the delusions, hallucinations, and illusions and the mania. Predisposition to insanity is not by any means an unfavorable symptom.

Diagnosis. Mania is to be differentiated from—

Toxic stimulation of alcohol, opium, cocaine, and cannabis indica;
Delirium grave;
Meningitis, of traumatic, cerebro-spinal, or aural origin;
Pyeluria;
Delirium from organic brain disease;
Post-febrile delirium from peritonitis, typhoid, typhus, scarlatina, rubeola, etc.;
Epileptic insanity;
Hysterical insanity;
General paresis;
Periodical insanity.

Toxic stimulation of alcohol, opium, cocaine, and cannabis indica or hasheesh should be readily differentiated by the history of the case.

Delirium grave can be distinguished by the greater violence of the patient, alternating with entire lucidity, and by the anæsthesia and self-mutilation that so often occur. The delirium of meningitis is usually mild in form, and attended by a hebetude not seen in mania, besides which there will be the history of the trauma, or the symp-

toms of cerebro-spinal meningitis, or an aural trouble precedent to the delirium.

It will sometimes happen that pyeluria will induce a vague, mild mania, which may be entirely misinterpreted if the urine is not examined. I have seen two such cases for which the certificates were filled out for committal to a lunatic asylum, but which recovered under ordinary treatment of the pyeluria.

The delirium of organic brain disease needs only to be alluded to.

Hallucinatory insanity following peritonitis, typhoid fever, typhus fever, scarlet fever, rubeola, etc., should not be confounded with idiopathic mania, as each has an entirely different prognosis, and the treatment is often very different.

It not infrequently happens that the periodicity of the mania incidental to a periodical insanity will be overlooked, and this should be borne in mind in any case of mania.

Epileptic and hysterical insanity need only to be mentioned.

It is possible that a general paresis may commence with symptoms of acute mania, and this should always be remembered. It is often impossible to make a diagnosis at first. A tremor of the tongue and facial muscles is often present in mania, and must not be confounded with that which may take place in general paresis. The safest rule is to wait for the lapse of a few weeks, and then the persistency of the facial and lingual tremor, a remitting irregularity of the pupil, and evident dementia, will make the diagnosis of general paresis extremely probable.

TREATMENT. The treatment of mania should be by means of the hydrobromate or hydrochlorate of hyoscine, bromide of potash, hypnotics when necessary, isolation, and attention to the general health.

The most effective drug at our disposal for controlling the restlessness of mania is the hydrobromate or hydrochlorate of hyoscine, which has the great advantage of not depressing, even when given in large and continued doses. The dose at first should be $\frac{1}{100}$ grain, in the form of tablet triturate. This should be given two or three times a day, or, if necessary, even every three or four hours. Bromide of potash, gr. 10 to 20, should be administered with it, the dose being given two or three times daily, as may be necessary. Usually in these cases of mania the insomnia is not profound, and can be met by the two drugs just mentioned and by attention to the nutrition. If, however, it should be necessary to employ hypnotics, the best is either sulphonal or chloralamid, the former in doses of 10 to 20 grains at bedtime in tablet triturates or in a powder given in a cup of chocolate, beef-tea, or milk; the latter in tablet triturate or elixir. If the patient should refuse to take medicines, they can be often given in food. The tablet triturates of hyoscine made for hypodermic purposes can be readily dissolved in milk or beef-tea. The bromide of potash can be sprinkled over the food just as ordinary salt is. The hypnotics can be dissolved in a cup of milk, beef-tea, or cocoa. Great attention should be paid in these cases of mania to the condition of the general health. Abundance of nourishing

food, especially large quantities of milk, should be regularly and systematically given. Alcoholic stimulants in moderate quantity should also be employed, best in the form of milk punch or egg-nog. Sedulous attention should be paid to the condition of the bowels and skin. These cases should be rigidly isolated, and they will always be best handled by trained nurses, for the reason that, like all the other insane, they are more controllable by strangers than by relatives. It is almost impossible to obtain any degree of rest in mania until the disease has been gotten well under control. At this period, however, it is always well to insist upon the patient lying in bed for at least twelve hours out of the twenty-four and resting two or three hours during the afternoon. The same attention should be paid to the menstrual period of women as has been advised in "Melancholia," and the same considerations will obtain about committal to a lunatic asylum.

CHAPTER IX.

KATATONIA.

Definition. Katatonia is a cerebral disease, with cyclical symptoms ranging in succession from primary melancholia to mania, stupidity, confusion, and dementia, one or more of these stages being occasionally absent, whilst convulsive and cataleptoid symptoms accompany the mental changes.

History. This remarkable disease, about which there has circled a considerable warfare, was first described by Kahlbaum in 1874, and to-day, twenty-one years afterward, alienists are hopelessly divided in opinion as to whether the author's claims to have discovered a new clinical type are warranted or not. As described by Kahlbaum, katatonia is a cyclical insanity, passing through five different stages respectively of melancholia, mania, stupor, and intellectual feebleness, with connected delusional conceptions that are slightly logical, often incoherent (confusional insanity), and, last of all, dementia. Each stage is extremely variable in duration, and in each may be observed alternating depression and excitement. Most frequently mania follows the initial melancholy, or melancholy with stupor may take the place of mania, and in cases of this latter kind it will be often found that there has been mania in preceding years. It may happen in rare instances that the melancholia with stupor is the first stage, occurring most frequently after violent physical or mental shocks, such as intense fear, traumatism, etc. In other cases, after a stupor of short duration, there is again a period of excitement or melancholy, which is again succeeded by the stupor, with or without mania. Occasionally, in the midst of a long stage of mania, lasting for a week or a month, there will be intermingled some days of stupor, or, more rarely still, the stupor alternates with a condition of confusional insanity. The terminations are in dementia, cure, or death. The different stages of melancholia, mania, stupor, intellectual enfeeblement, and dementia offer nothing special in their symptoms; but the stage of exaltation, whatever it may be, has certain characteristics, being either of the type of melancholia agitata, or most violent excitement, or an insanity with logical and fixed delusions that we should nowadays call paranoia. There are other symptoms that Kahlbaum claims as peculiar to katatonia. First, there is a theatrical, so-called pathetic manner of the patient, often as of a person declaiming, or of one acting a tragical or religious part, the patient declaiming or reciting constantly, with gesticulations, but incoherent in all that is said. Secondly, there is a tendency to the repetition of words and phrases without sense or connection, but pronounced as if the patient were speaking in public,

and to this symptom Kahlbaum has given the name of *verbigeration.* This verbigeration may, in the course of the disease, pass gradually into the silly loquacity of the ordinary dement or the rapid succession of ideas of mania. Thirdly, there is often observed a remarkable tendency to make use of diminutives, of which, it may be remarked, in passing, the German language contains a larger quantity than any other modern tongue. With these mental symptoms are associated certain marked physical ones. Often in the stupor, less frequently in the other stages, there is a cataleptic condition of the extremities, and convulsions are frequently observed of a choreiform, epileptiform, or tetaniform nature. There is never any motor paralysis, but occasionally some anæsthesia has been observed, whether apparent or real is a question, and also frequently hyperæsthesia, while localized occipital headache is said by Kahlbaum to be characteristic. Among other symptoms of inferior diagnostic value that have been noted by Kahlbaum may be mentioned œdema of the lower extremities, occasionally of the lids, cutaneous desquamation, anorexia, fetid breath, constipation, and chlorosis. Phthsis is very frequent in this disease, although Kahlbaum has found it rare in other forms of mental disorder, and it is probably for this reason that pneumonia is more frequent in the fatal cases than it is in general paralysis.

The list of causes that have been offered by Kahlbaum throws no light upon the disease at all, and consists only of such etiological factors as are found in almost every serious affection. Kahlbaum claims that there have been epidemics of katatonia, and that the so-called *convulsionnaires* were katatoniacs; but as these mental or hysterical epidemics are claimed by the votaries of so many other diseases, we may well pass over this assertion in silence.

As regards the pathology, Kahlbaum institutes a comparison with general paralysis. He believes that in the onset there is a condition of stasis in all cerebral vessels, with serous exudation, leading to softening of the cerebral tissue without retraction, but with formation of exudations upon the interior surface and upon the membranes, particularly at the base. In the more advanced cases there are a retraction of tissue and an organization of the exudates. The arachnoid is always affected, particularly where it stretches from the pons to the medulla oblongata behind the fourth ventricle, although the exudation may extend along this same membrane upward to the fissure of Sylvius and the second and third frontal convolutions, which Kahlbaum would have us regard as a possible explanation of the mutism observed in the stage of stupor or of the verbigeration. Pulmonary and intestinal tuberculosis are very constant accompaniments.

Upon the publication of Kahlbaum's memoir, the question at once arose as to whether a new clinical entity had been discovered, or whether a description had been given of different forms of insanity complicated with cataleptic and convulsive symptoms. Almost every large meeting of alienists in Germany has had this question before it for solution, and a formidable literature upon this subject has

accumulated. In support of Kahlbaum's claims are ranged Hecker, Brosius, Neisser, Schüle, and Neuendorf, in Germany; Etoc-Demazy, in France; Spitzka, Hammond, and Kiernan, in America. Those denying Kahlbaum's claims are Arndt, Westphal, Tigges, Von Reinecke, Krafft-Ebing, and Tamburini. The opponents of Kahlbaum maintain that there is no such form of disease as he describes under the name of katatonia or insanity of tension (*Spannungs-Irresein*), or that the main symptoms are found in other forms of insanity. Fink has reported three cases of hebephrenia closely resembling katatonia. Krafft-Ebing and Ritti range this disease under the head of circular insanity. Tamburini has cited a number of cases of melancholia attonita with cataleptic symptoms; and a large number of observations of melancholia with stupor complicated by cataleptic symptoms have been published by Angelucci, Wigglesworth, Voisin, and Wagner. There can be no question that the somatic symptoms in katatonia, upon which Kahlbaum lays so much stress, are found in many other forms of insanity, and hysterical neuro-muscular hyper-excitability is very frequent in catalepsy and in choreiform, tetaniform, and epileptiform convulsions. Catalepsy has been described also by Morel and Séglas in paranoiacs, imbeciles, epileptics, and in melancholia, with or without stupor. Guislain and Morel had observed ecstasy in many diseases, particularly hysteria, long before Kahlbaum wrote; and others have seen it in melancholia, in circular insanity, in alcoholic intoxication, hypochondria, and mania. Séglas has reported contracture in general paresis, whilst Knecht has observed a number of the other katatoniac symptoms in this latter disease, in which Sage has also noted choreiform movements. The verbigeration has been observed by Vogelgesang and Jastrowitz in general paralysis; the stereotyped gestures by Brugia and Maezocchi in the same disease, with or without delusion; and Morel's celebrated *gémisseur*, or sigher, clasping his clothing convulsively with his left hand, whilst his right arm kept rhythmical time with his right foot tapping the floor, was a notable example of this kind of movement, although he was evidently a case of paranoia. The eccentric and declamatory attitudes are extremely frequent, as any alienist knows, in many forms of insanity.

From all this it will be seen that the question is as yet under discussion, and that only a very small number of alienists support the claims of Kahlbaum.

The treatment of katatonia is not understood, and therefor a case that seems to be of this kind should be treated upon the same principles as those indicated in the sections respectively upon "Mania" and "Melancholia."

CHAPTER X.

PERIODICAL INSANITY.

THE laws of periodicity which obtain among all natural phenomena are, as we should naturally expect, present as well in insanity; and to the oldest physicians certain periodicities were well known. As far back as 1791 Pinel wrote upon periodic or intermittent mania. The alternations of chronic forms of insanity are every-day matters to hospital attendants, so that orderlies are familiar with the good and bad days of patients; whilst every general practitioner knows the periodical influence of menstruation upon mental characteristics. Indeed, it may be said that almost all the phenomena of health and disease have a periodicity that is exemplified in sleep, appetite, difference of feelings in the spring, autumn, and winter, periodical returns of certain diseases such as dipsomania, malaria, occasionally neuralgia, especially migraine; and I have seen as marked periodicity in cases of intracranial syphilis as in malaria. It is probable that the ancient notion of the moon affecting the insane was due to this observance of periodicity in mental disorders, so that from this idea may have been derived the term *lunacy;* but the first account of periodical insanity as a distinct form of mental disease was given by Falret and Baillarger in 1854. Curiously enough, by one of those singular coincidences which occur so often in the history of medicine, the description of the same disease was given at the same time by two men, each seeming to have been perfectly honest in his publication and without knowledge of the other's researches. Emmerich claims that Griesinger was the first to observe this form of insanity, and that he described it in his book in 1845; but he gave it no name, speaking of it merely in passing, so that the credit of the discovery undoubtedly belongs to the two Frenchmen. Baillarger termed it *folie à double forme* or *formes alternes*, whilst Falret denominated it *folie circulaire.* Baillarger again refers to the subject in 1880. These two authors describe a condition of mania followed by melancholia; but there seems to have been some difference between the two as to whether or not there was a lucid interval between the two conditions, or whether the melancholia immediately followed the mania. However, it is undoubtedly true, as is said by Falret's son, writing in 1890, that this lucid interval may or may not be present. The essential distinction is that these two conditions of mania and melancholia respectively follow each other, and whether there is a lucid interval or not is immaterial. The different stages vary greatly, from months to years, and so may the intervals, and even in the same attack the different stages will not be of corresponding length.

The Germans call these cases *Zirkelwahnsin* or *zusammengersetzte Psychosen*, whilst they have been called by the French *manie melancholique.* Wittkowski described what he called *recurrirende Manie* or recurrent mania, an unfortunate name, as it does not serve the purpose of designating the special form that he detailed, which consisted of two attacks of mania separated by a lucid interval. He had seen only five cases among one thousand patients, and they ran in this order: 1. Fifteen days of the first attack, thirty days interval, thirty days of the second attack. 2. Seven days of the first attack, fourteen days interval, fifteen days of the second attack. 3. Four days of the first attack, about one month lucid interval, and about five weeks of the second attack. 4. Nine days of the first attack, thirty-one days lucid interval, eight days of the second attack. 5. Ten days of the first attack, eleven days lucid interval, nine days of the second attack. Recurring attacks of mania and recurrent attacks of melancholia have been frequently described, but of all the cases on record none has been erected into distinct types except those that have been described by Baillarger, Falret, and Wittkowski. Classifying cases of periodical insanity, therefore, we may say that there are:

The circular insanity of Baillarger and Falret;
The recurrent mania of Wittkowski;
Ordinary mania, or melancholia, with periodicity.

In the circular insanity of Baillarger and Falret neither the mania nor the melancholia is typical, the mania being rather a condition of excitation and garrulousness of the degree that constitutes high spirits or that would denote great eccentricity, whilst the melancholia is a condition of depression and low spirits, and not of the anxiety, restlessness, insomnia, post-cervical ache, and suicidal tendency of true melancholia. Some of the cases of circular insanity will do the most remarkable things in the condition of mania, perform the most eccentric acts, or manifest the most erotic tendencies. Thus, one patient of mine always became very restless, would go out on the street and amuse himself by pitching coins to newsboys and watching them scramble, was exceedingly gallant, and offered himself to a half-dozen women, telling the most marvellous Munchausen yarns to blind their fancy, becoming witty, loquacious, and absolutely irrepressible. In the melancholiac stage all this is reversed, and the patient becomes taciturn, sluggish in movement, moping, inattentive, listless. The bodily weight increases in the stage of mania and decreases in the stage of melancholia, and the appetite follows the same law. Indeed, all the bodily functions are greatly increased in the stage of mania and greatly depressed in the stage of melancholia, so that the flushed face and increased bodily heat of the former stage merge into the cold extremities and waxy skin of the latter. The lucid interval of circular insanity, in my experience, is not a true lucid interval. The patient is rather in a state of remission than of absolute intermission, although the slight difference may only be seen by one accustomed to deal with cases of insanity, or by some intel-

ligent member of the family who can notice the difference in mentality.

These cases of circular insanity are of very evil prognosis, so far as complete cure of the malady is concerned, although it is sometimes possible to cut short a single attack.

The recurrent mania of Wittowski is a very rare disease, and I myself have never seen a case of it. All of Wittowski's cases, however, recovered, but he does not state whether they had further recurrences of the trouble.

Ordinary recurrent mania and recurrent melancholia will have a doubtful prognosis, although not necessarily a hopeless one, and of these the melancholias have the best prognosis. Clouston is of the opinion that the periodicity of mental disease is one of the signs of an educated race that is dying out, or, in other words, that periodicity occurs mainly in what the French call the degenerated. He observed 40 to 46 per cent. of the periodical cases among 338 of various forms of insanity, and of these one-half had monthly periodical changes, one-third were somewhat irregularly seized, and the remaining one-sixth were entirely irregular.

CAUSATION. No special cause has ever been determined for periodical insanity, over and above the general causes of mental disease.

DIAGNOSIS. There is usually no difficulty in recognizing circular insanity with its successive stages of mania and melancholia, one either following the other or separated by a so-called lucid interval, nor should there be any difficulty in recognizing Wittkowski's recurrent mania, or the other forms of less sharply marked periodical insanity, when these diseases have gone through their different stages of development; but the danger usually is that they may not be recognized at the start, or that some other form of insanity may be mistaken for them. The initial mania of circular insanity has more of eccentricity and odd high spirits about it than are found in ordinary mania; and in the latter, delusions, hallucinations, and illusions are far more frequent, so that a gradually developing mania that is more like eccentricity than downright insanity should always put us on our guard. The stage of melancholia following the mania is sometimes not recognized, because it is thought to be the necessary depression which precedes convalescence in many acute or subacute insanities; but the diagnosis of periodical insanity should especially be made from epileptic insanity, because as good an observer as Doutrebente was deceived by a case of larvated epilepsy, and diagnosed it as circular insanity.

TREATMENT. Some cases of periodical insanity, even of circular insanity, can be very materially benefited by treatment so far as regards the individual attack. Thus, several authors have spoken—and I have myself seen it do good—of the excellent effect in the mania of circular insanity, or in the recurrent forms, of cannabis indica, the tincture, in ♏xv–ʒj doses, as often as may be necessary, either two or three times a day, or every few hours. To this may be added with great benefit the bromide of potash in drachm doses. The sulphate of quinine in large doses, 10 to 60 grains a day, is also

often of considerable benefit, and Baillarger has seen excellent effects from its continued use for days in these large doses. I can confirm this of my own experience, although I have sometimes had to administer the bromide of potash with it because of the cinchonism or the general nervousness that it induced. Hyoscine, either the hydrochlorate or the hydrobromate, is also an excellent remedy in doses of $\frac{1}{100}$ grain twice, thrice, or four times a day. The stage of melancholia cannot be treated so well as the stage of mania; indeed, I think that the treatment of periodical insanity should be the vigorous treatment of the stage of mania with the hope of cutting it short. The stage of melancholia can sometimes be influenced, however, by the use of opium in the form of the aqueous extract, $\frac{1}{8}$ grain once, twice, or three times a day, or codeia, in conjunction with the sulphate of strychnine, $\frac{1}{50}$ to $\frac{1}{30}$ grain, three times a day, or the sulphate or tannate of quinine in tonic doses. Both of these can be given in the form of tablet triturates; and if the patient refuses to take medicine, the hypodermic tablet triturates can be dissolved in food, such as milk, beef-tea, or cocoa.

CHAPTER XI.

FUROR TRANSITORIUS.

Definition. Furor transitorius is an outbreak of violent fury, lasting for a few hours, and terminating in deep sleep, from which the patient awakes without the slightest memory of what has occurred; and never recurring in the same person.

Clinical History. Transitory fury has occasionally vague and short prodromal symptoms, but these are more frequently entirely absent. When they do occur, they consist of restlessness, irritability, flushed face, headache, or a sense of pressure and beating in the head, vague general sensations, malaise, cardiac irregularity, vertigo, perspiration, and roaring in the ears. They usually last only for a few minutes before the attack begins. The onset of this is sudden, and it is characterized by the blindest fury, the patient smashing articles of furniture and tearing his own clothes into bits, at the same time that he howls or growls or murmurs inarticulately, This attack may last a few minutes or a few hours, when the patient suddenly becomes quiet, and passes into a deep sleep of hours' duration, from which he awakens without the slightest memory of what has taken place.

Causation. The causes of transitory furor are extremely uncertain, but it has been known to occur after mental or physical overexertion, in the puerperal state, from rapid changes of temperature in the room or the atmosphere, with indigestion and gastric disturbances, and also from the inhalation of carbonic acid gas from an overheated stove.

Diagnosis. The diagnosis will be principally from transitory mania and epileptic mania, and for this I would refer my readers to the section on diagnosis in "Epileptic Insanity."

Prognosis. The prognosis, so far as the recurrence of the attack is concerned, is excellent, because it has never been known to recur in the same person.

Treatment. Treatment can seldom be brought to bear, because the attack is over before the physician has been called or, the patient is so violent as to render it utterly impossible to administer any medicine; whilst, on the other hand, it is of course useless to do anything when the patient has passed into the terminal slumber. In case of necessity, however, a hypodermic injection of $\frac{1}{50}$ to $\frac{1}{25}$ grain of Merck's hyoscyamine might be of excellent effect, I should think, although I have hitherto never had an opportunity of essaying it.

CHAPTER XII.

EPILEPTIC INSANITY.

Definition. Epileptic insanity is the insanity occurring in epileptic individuals before the attacks, during them, after them, between them, or taking their place.

History. Although insanity in the epileptic has been known as long as epilepsy itself, and although such older writers as Esquirol, Pinel, Brach, Garard, and Hall, have described epileptic insanity with some degree of precision, it is yet true that the first really precise study of the subject was made by Falret in 1861; but it was not until 1875 that the first exhaustive paper appeared, by Samt; whilst Hughlings-Jackson in the same year elucidated the matter with a philosophic catholicity that needed only fuller clinical detail to have made it complete. Since this time a number of writers have busied themselves with this topic; but, with the exception of certain medico-legal details, nothing has been added to the clinical disquisitions of Falret and Samt.

Clinical History. Although Samt enthusiastically advanced the claim that epileptic insanity had certain features of its own by which it could be recognized without any history of antecedent epilepsy in the patient, his views have yet met with no support from alienists, and, highly desirable as it might be for medico-legal considerations to be able to diagnose epileptic insanity *per se*, the fact still remains that this cannot be done, as in epileptics the following forms of mental disorder have been described:

- Falret's intellectual *petit mal* and *grand mal;*
- Stupor, with or without verbigeration and alliteration;
- Circular insanity;
- Paranoia;
- So-called larvated insanity, or insanity taking the place of the usual epileptic attacks;
- Chronic insanity;
- Dementia.

In all these different types there are certain general characteristics, which, together with the history of the epilepsy, serve to demarcate epileptic insanities from the non-epileptic varieties, although, as has been said, they are not absolutely conclusive, merely serving as what the lawyers call *prima facie* evidence; *i. e.*, evidence which points a certain way, but which other facts must confirm or refute. These characteristics are: the epileptic *facies* and character; a strong tendency to automatism, dreaminess, violent outbreaks, seemingly erratic losses of memory, and sudden recoveries. The epileptic *facies* is difficult to describe, although it can usually be readily detected by

one accustomed to dealing with this class of patients; but, as nearly as it can be put into words, it consists of a commingled suspicion, slight dreaminess, and moroseness. The automatic acts of the epileptic are akin to those of the somnambulist—indeed, often surpassing those of the latter; and this fact is of very great medico-legal importance. I have known an epileptic to roam about the city for days, seemingly acting with perfect intelligence, so that no suspicion of his real condition was excited, and yet returning to his normal consciousness either without any remembrance of where he had been or what he had been doing during this time, or with only fragmentary recollection of the period. Many instances of this kind are upon record. Other epileptics may commit crime with deliberation, from motive and with cunning, and Echeverria has collected many such cases. Most cases of epileptic insanity will have a period of dreaminess in some stage of the mental manifestations, usually in conjunction with delusions, and with this is frequently associated the condition for which Kahlbaum has suggested the name of *verbigeration;* whilst sometimes a curious alliterative repetition of words is observed, one patient thus repeating: "I have tum, tum, tum, tum, del, del, del, tumbled and delivered——."

Violent outbreaks are exceedingly common in the different forms of epileptic insanity, and it is an axiom with alienists that these cases are, next to those of transitory fury, the most dangerous of all classes of lunatics. The losses of memory in epileptic insanity are very curious, and have led to some terrible mistakes medico-legally, having been too often misinterpreted in the courts. The loss of memory may be entire, partial, or complete for a time, with entire restoration. Thus, a patient may have absolutely no remembrance of what he has done at a certain period when he had perhaps committed a crime, or he may forget entirely for a time and then afterward have a complete remembrance of the period, or he may have only a partial remembrance restored to him, or, what is still more confusing, his partial or entire restoration to memory may again be lost. Samt gives some remarkable illustrations of these facts. Another curious characteristic of the epileptic memory is that a patient losing consciousness by becoming epileptic, from any of the numerous causes, may be deprived of memory absolutely for all that has occurred after the loss of consciousness, and then, coming to himself, may have his memory sharply resumed from the time when he lost it. Thus, cases of *petit mal* may stop in the middle of a sentence, pass through their attack, and, coming to themselves, go on with the sentence from the point where they left it off. Hunter McGuire, of Richmond, Va., details a most remarkable case of this sort, of a negro who had been wounded in the battle of Manassas by a splinter of shell fracturing the inner table of the skull. He became an epileptic, and remained such for some ten years, when Dr. McGuire operated upon him, removing the splintered inner table, and the first words that he uttered on recovering from the ether were: "Where is the —— regiment?" thus displaying the absolute lack of memory for everything that had occurred from the time of his

wounding. Sudden recoveries often occur in epileptic insanity, so that a patient who has been violent and uncontrollable one day may be perfectly rational upon the next. This is a third fact of grand medico-legal value.

The intellectual *petit mal* of Falret consists of confusion of ideas, with instantaneous instinctive acts that are often violent. These cases generally begin with sadness or moroseness without cause, and then become silent, irritable, and confused. They are semi-conscious, of confused memory, and it is difficult to enlist their attention. They often leave home suddenly and wander aimlessly. They consider themselves wretchedly unhappy, often think that they are persecuted by their families or friends, and may accuse others of being the cause of their anxieties or their torments. Any dislike or hatred to an individual which they may have had in their normal condition may now become greatly aggravated, and under the influence of some such motive, or without it, they may be guilty of suicide or some such crime as theft, arson, or homicide, or may do some violent impulsive act, such as butting their heads against the wall, smashing things, or venting their rage upon some innocent individual. After any one of these violent acts the patient usually comes to himself, or this may happen without any such precedent violence. This condition of *petit mal* varies in duration from a few hours to several days, and may recur again and again. The intellectual *grand mal* of Falret was known to the older French writers as mania with fury (*manie avec fureur*). The onset is usually brusque, although it may sometimes have certain prodromata, such as cephalalgia, vomiting, flushing of the face, brilliancy of the eyes, alteration of the voice, slight convulsive movements of the face or extremities, or sadness, irritability, and slight excitation. These prodromata last but a few hours. Another curious feature of this form is that one attack always closely resembles every future attack in the same subject. The mania is one of the greatest violence and brutality, so that the patient is very destructive to anything and everything about him, at the same time that he is possessed of terrifying and lively delusions and marked hallucinations of hearing, smell, and especially of sight. The hallucinations of sight are very generally of some red color. There is a marked incoherence, however, in the delusions and hallucinations, which succeed one another with great rapidity. These attacks seldom last more than a few days, and cease as suddenly as they have begun, the patients usually returning instantaneously to their normal condition, although sometimes there may be a short consecutive period of slight stupor. As has been well said by Falret, they awake as from a horrible nightmare.

The stupor of the epileptic is generally a dreamy, delirious condition, with occasional automatic acts of violence, and with verbigeration and alliteration such as have been already spoken of.

Circular insanity has also been described in epileptics; but it is an exceedingly rare condition, and many cases of so-called circular insanity in the epileptic have been simply the intellectual *grand mal* of Falret, succeeded by more profound stupor than usual.

Gnauck has observed a spurious form of paranoia to occur in epileptics; but his cases and the few which I have seen should really be called *paranoioid insanity*, inasmuch as they have a much shorter duration than the ordinary cases of paranoia, whilst the ideas of persecution and self-exaltation are less defined than in the latter disease.

The so-called larvated insanity of Morel was claimed by him to be a form of epilepsy of the type of intellectual *grand mal*, taking the place of the regular epileptic paroxysms as a sort of equivalent. Hughlings-Jackson, however, believes that all these are preceded by attacks of ordinary *petit mal* which have been overlooked, and he cites cases to prove his assertion. It should be remembered that *petit mal* may consist of merely so slight a loss of consciousness as to constitute scarcely more than a vertigo, either without any muscular movements, or with very slight twitchings of the facial muscles, so that an attack might very readily be overlooked even by the patient's family or those immediately surrounding him. It is well to know, however, that such cases of mania do occur in epilepsy, and that the name of *larvated* or concealed in an excellent one, even though they are preceded by such slight attacks of *petit mal.* These larvated attacks of epileptic insanity are apt to recur periodically or quasi-periodically, and they have in several instances been mistaken for periodical mania.

Whilst most cases of epileptic insanity are temporary in duration, varying from a few hours to a few days or weeks, the insanity may also become chronic and last for years.

Dementia is the usual termination of most cases of severe organic epilepsy, or of frequently recurring different forms of epileptic insanity; but I think that the usual statements as to the inevitable tendency of cases of epilepsy to dementia are very much exaggerated. As has been said in the chapter upon "Epilepsy," epilepsy is only a symptom, and there are many forms which do not tend to true epilepsy at all, such as the nutritional, the laryngeal, the form associated with migraine, and many cases which recur infrequently.

The medico-legal responsibility of the epileptic has been a question about which a great deal of confused thinking has been done. It is monstrous to assert that a person is not responsible for his or her acts because he or she is subject to epilepsy. On the other hand, it is equally monstrous to assert that an act done under the influence of epilepsy should be punished by the law because there has been evidence of deliberation and intelligence about it, or because it has been done with a motive. The simple questions in any given case are:

1st. Was the individual in question suffering from some one of the many forms of epileptic insanity? or,

2d. Had the individual in question had an epileptic attack at such a time before or after the act committed as to render it probable that he or she was at the time of the committal of the act under the influence of epilepsy?

In either one of these events the individual in question is undoubtedly irresponsible, never mind how great may have been the seeming

deliberation and intelligence or the motive. It should always be remembered, as has already been said, that the epileptic individual may have the most curious and seemingly contradictory losses of memory, so that he may remember an act immediately after it has been committed, lose this memory later, and regain it still later on. All epileptic criminals, however, are dangerous, and society should be protected against them, not by their incarceration in a prison, but by their isolation in an insane asylum.

CAUSATION. The causes of epileptic insanity are the causes of epilepsy which have been narrated at length in the chapter upon that subject.

DIAGNOSIS. When a patient has had an attack of epilepsy, either of the type of *petit mal* or *grand mal*, and this is succeeded or preceded by mental derangement, the diagnosis is easy enough; but more care is required in reaching a conclusion about the nature of an insanity or a criminal act of an individual who is merely known to be subject to epilepsy, but in whom the insanity or criminal act has not been preceded or succeeded immediately by an epileptic manifestation. In such a case the criteria which have been mentioned will become of great diagnostic value, namely: the epileptic facies and character, the tendency to automatism, the dreaminess, the violent outbreaks, the seemingly erratic losses of memory, and the sudden recoveries. The claim made by Samt, however, that mental aberrations with these characteristics can be positively diagnosed as epileptic, even though the patient has had no attack of *petit* or *grand mal*, has not been adopted by alienists in the twenty-one years that have elapsed since Samt's publication, for the very good reason that non-epileptic insanities will sometimes, though rarely, have these features.

The diagnosis of epileptic insanity should be from—

Transitory frenzy;
Acute mania;
Periodical insanity;
Hallucinatory insanity;
Paranoia.

The patient subject to transitory frenzy has no epileptic manifestations of any kind. The attack has never been known to recur; indeed, the individuals subject to it rarely manifest any symptoms of insanity at any other time in their life.

The acute mania of the non-epileptic is far less violent, far less characterized by brutality, and much more marked by rapid and changing ideation, generally of a beneficent character, so that when any violence is done, it is because of irritation or opposition, whilst the onset is not so apt to be sudden, and the convalescence is much more gradual.

Periodical insanity is sometimes mistaken for epileptic insanity; but the diagnosis can be made by the regular recurrence without a history of epilepsy, the far less tendency to automatism and violence, dreaminess, and loss of memory, and the alternation of mania and melancholia, with or without a so-called lucid interval in the

form known as circular insanity. On the other hand, recurring epileptic insanity should not be mistaken for periodical insanity, and the diagnosis can be made by the absence of a history of epilepsy.

In hallucinatory insanity the diagnosis will be made by the predominance of the hallucinations and delusions, and by the lack of the history of epilepsy and of the epileptic characteristics.

Paranoia presents an entirely different picture from that of epilepsy, although Gnauck has reported so-called cases of paranoia in epileptics; but, as has been said, they differ essentially from the true paranoiacs, inasmuch as the delusions of persecution and self-exaltation are confused and intermingled with the peculiar lapses of memory, epilepsy, and the epileptic mental characteristics.

Treatment. The bromides do not have the same influence upon the insane manifestions of epilepsy as they do upon the non-insane symptoms. The best drug is usually hyoscine, the hydrobromate or the hydrochlorate, in doses of $\frac{1}{100}$ grain, once in one, two, or three hours, best given hypodermically. Cannabis indica is also a very useful medicine, and the best preparation to use is the fluid extract in doses of 5 to 10 drops every hour or two until the sample has been tested. In some cases the application of an ice-bag to the spinal column for an hour or two at a time has broken short an attack. Occasionally brisk purgation with elaterium is of value, using doses of $\frac{1}{4}$ grain every two or three hours until the effect is obtained ; or, if the patient's condition be such that this may act as too much of a depressant, a brisk saline will answer the purpose. I have also known an attack to be broken up by a powerful counter-irritant, such as a large blister somewhere about the body, or the application of the actual cautery to the back. In the more chronic forms of epileptic insanity cannabis indica, in combination with the bromides (*vide* Chapter IX., under "Epilepsy"), will be found the best treatment.

CHAPTER XIII.

DELIRIUM GRAVE.

Synonyms: Mania gravis. Phrenitis. Typhomania. Acute delirium.

Delirium grave is a very rare disease, characterized by furious outbreaks of maniacal excitement and violence, with intermissions. The patient is extremely incoherent, breaks, kicks, screams, talks incessantly, has hallucinations that are usually fixed and often very vivid, and there is generally such anæsthesia that injuries are not felt, so that one patient actually gnawed off an ungual phalanx of a finger. Great insomnia is usually present; the temperature ranges from 100° to 103°, sometimes even higher; the pulse is frequent and soft; and there is rapid emaciation. To this excited period usually succeeds one of depression, almost of collapse, in which the patient lies quiet, staring, muttering, mute, as in the terminal period of acute exhaustive diseases. These conditions of excitement and depression may alternate, and in some cases the depression may not be extreme, so that the patient may be aware of his condition. The disease almost always terminates fatally, and when recovery does occur it is incomplete, leaving some permanent aberration of mind or passing gradually on into general paresis or some form of dementia.

In cases of delirium grave the pathological lesions are: great cerebral and spinal hyperæmia, often with œdema; and according to Spitzka, the cortical ganglion-cells are granular or opaque, stain poorly, and their peri-ganglionic spaces, like the adventitial lymph-sheaths, are literally crammed with the formed elements of the blood. This hyperæmic congestion has been supposed to be due to a vasomotor paresis; but this, like most of the vasomotor theories, is the purest kind of hypothesis.

The diagnosis of delirium grave should be from—

Acute mania;
Meningitis;
Transitory furor;
Agitated melancholia.

In acute mania the patient does not have the anæsthesia, and is not so wildly and unreasoningly furious, nor has he the intermissions.

In meningitis the onset is more gradual, the maniacal outbreaks are not nearly so furious, the delirium is more apt to be muttering or mildly vivacious, and there is often a characteristic retraction of the head.

In transitory frenzy the outbreak is more sudden, more like an epileptic attack, lasts only a few hours instead of a few weeks, and

the patient is perfectly himself again when the attack is over; so that there is no similarity between this disease and delirium grave, except in the one symptom of the maniacal outbreak.

Agitated melancholia should never be confounded with delirium grave, because the agitation is not a furious maniacal outbreak, but is a restless, impulsive act or series of acts in a very melancholy, depressed, often suicidal individual. I refer to the differential diagnosis in this place only because I have seen several mistakes made.

The prognosis of delirium grave is exceedingly grave, as there are very few recorded cases that have not terminated fatally.

The only treatment for delirium grave that has been of any use has been that claimed by one writer, with large doses of ergot, which should be given in the form of the fluid extract, in doses of a drachm every two hours. Bromide of potash in doses of 10 or 20 grains should also be given every two or three hours, with hydrobromate of hyoscine, $\frac{1}{100}$ grain, at the same intervals during the excited stage. During the depressed stage the bromide and hyoscine should be omitted entirely. I believe, also, from my experience in a few cases, that large doses of the sulphate of quinine will be found to be of great value, and I should give 20 to 30 grains in the twenty-four hours, in the form of the tablet triturates or capsule, or, if the patient is averse to taking medicine, in solution with an acid; or the hydrochlorate of quinine, hypodermically, as in the following solution recommended by Hare:

℞.—Quin. hydrochlorat.		gr. vij.
Glycerinæ Aquæ destillat.	āā	f ℥ss.

M. ft. sol.
S.—Warm solution before using it, and do not add acid.

CHAPTER XIV.

HALLUCINATORY INSANITY.

Synonyms: Primäre Verrücktheit (Westphal). Hallucinatorische Verwirrtheit. Mania Hallucinatoria (Mendel). Delusional Stupor (Newington). Hallucinatorischer Wahnsinn.

DEFINITION. Hallucinatory insanity is a form characterized by quickly changing delusions, hallucinations, and illusions, with confusion of thought and alternations of excitement, depression, and stupor.

CLINICAL HISTORY. In hallucinatory insanity the patient's delusions, hallucinations, and illusions are constantly and quickly changed, and they are sometimes of a depressed or even terrifying nature, sometimes gay, sometimes vague in character. But the characteristic of them is a thronging ideation, so to speak, a constantly changing phantasmagoria of mental impressions, without logical connecting link between them, and the delusions are not therefore logical or *systematized.* The patient, however, may be calm and seemingly intelligent, although this calmness is very readily disturbed by the nature of the hallucinations, illusions, and delusions. There is a degree of intelligence, on the one hand, that is far greater than is witnessed in mania; whilst, on the other hand, the hallucinations, illusions, and delusions are infinitely more vivid and shifting in character. These patients, when the disease is not far advanced, can give very accurate and vivid accounts of their hallucinations and delusions, of which they retain an accurate memory; but in the severer types of the disease there are successive conditions of excitement and stupor from exhaustion and speechlessness, and of these the patient will often afterward have extremely defective memory. In some cases convulsions may occur, although these are infrequent. The convalescence is generally preceded by a mild form of dementia, although the patient may recover without this and by a gradual diminution of the other symptoms.

ETIOLOGY. The causes of hallucinatory insanity are—

Neurasthenia;
Anæmia;
Alcoholic and sexual excesses;
Many febrile diseases;
The puerperal condition.

Much has been said of the febrile and the puerperal insanities, but the most of them belong to this form, although the puerperal condition may produce also a pure melancholia or a pure mania. Peritonitis in the female is especially apt to be followed by hallucinatory insanity.

DURATION. The average duration is five to six months, although certain cases may run their course in a few days. The post-febrile and the menstrual are the shortest in duration, whilst the puerperal cases vary very greatly.

PROGNOSIS. Prognosis is favorable, if the patients can be subjected to proper treatment, and if it is commenced early enough. Exhausted individuals, however, or those who have had previous attacks, may pass into dementia or die.

DIAGNOSIS. The diagnosis of hallucinatory insanity is from—

Mania;
Melancholia;
Paranoia;
The mania of general paresis;
Delirium tremens.

In mania the hallucinations, delusions, and illusions are not nearly so prominent, the element of confusion is far less, and the latter does not come in spells lasting for hours or even a day or two, as in hallucinatory insanity.

In melancholia there is profound melancholy, stupor, or melancholy agitation, not the thronging ideation, hallucinations, illusions, and delusions of hallucinatory insanity.

Paranoia is characterized by logical delusions, great intelligence, and generally by calmness of manner; and confused hallucinations and delusions are only present in the exacerbations.

The mania of general paresis can be recognized by the history of the case and by the accompanying physical symptoms of tremor, defective speech, perhaps also by the peculiar gait, and the pupillary abnormalities.

In delirium tremens the history of the case, the hallucinations of sight and hearing, and often the great excitement, will make the diagnosis.

TREATMENT. The treatment of these cases of hallucinatory insanity should be by means of chalybeates, quinine, bitter tonics, abundant nourishment, and either alcoholic stimulation or the malt-extracts. Not one grain of a depressing remedy should be used unless it is absolutely necessary. It is my custom to see that these patients obtain large quantities of food at once, which they especially need because of the impossibility in most cases of keeping them at rest, even in a chair or on a lounge. I therefore satisfy myself that they really have three full meals a day, from one to two quarts of milk in the twenty-four hours, and a pound of beef made into a strong beef-tea and divided into three equal portions to be taken in the twenty-four hours. (*Vide* "Neurasthenia.") A careful attention should prevent these patients from becoming constipated, more especially when taking large quantities of food. The sleeplessness, which is apt to be an annoying feature, can be overcome by means of a milk punch or eggnog at bedtime. If this will not answer, 10 grains of sulphonal should be administered, with 10 grains of bromide of potash, at bedtime in beef-tea or cocoa or milk. If, whilst taking large quantities of food, the patient still remains excited and unruly,

$\frac{1}{100}$ grain hydrochlorate or hydrobromate of hyoscine should be given once or twice a day, and continued, if necessary, day by day. Hyoscyamine is a dangerous drug to use in hallucinatory insanity because of its depressing effect, and it should therefore seldom be administered unless the violence of the patient should make it necessary, when grain $\frac{1}{100}$ of it should be combined with 10 to 15 grains of potassium bromide, and increased as circumstances may require. Opium in some cases will have a very beneficial effect, but it is an extremely unreliable drug in this affection; nevertheless every case will be a guide to itself, and if the medicine is not found to produce improvement it should be dropped. The aqueous extract is the best preparation to use, and $\frac{1}{8}$ grain may be given once or twice a day. It is often extremely difficult to get these patients to take medicine, through perversity or some delusion. If this be so, the soluble triturates of opium and hyoscyamine should be used; and in giving iron the carbonate of iron can be mixed with tea, beef-tea, cocoa, or gravy, a drachm being given three times a day in this manner, as it is nearly tasteless. Quinine and the bitter tonics are often very difficult indeed to administer, although the tannate of quinine will sometimes be taken in the form of chocolate lozenges.

CHAPTER XV.

DEMENTIA.

THERE has been considerable diversity of opinion among alienists as to the clinical forms of dementia and their prognosis. Dementia has come to mean technically the same thing as stupidity, and it is called by this latter name by the French authors. It may be clinically divided into two great types, namely :

1. Primary dementia;
2. Secondary dementia.

The primary dementias may be due to exhaustion of the brain, to psychical shock, and to mechanical shock.

That which results from exhaustion of the brain varies greatly in degree. It is what some authors term cerebral asthenia or neurasthenia. In the slighter forms it may consist of a simple defect in memory showing itself occasionally, and an inability to concentrate the attention upon mental problems. Thus, the patient will find that he cannot remember names, events, or faces, and that his brain refuses to functionate in those delicate mental processes which we call thought. He may thus read over and over again a page that he would ordinarily have no difficulty whatever in mastering, and yet be unable to rise from its perusal with a knowledge of what he has read, much less with a mental assimilation of it. It is the chronic analogue of what all feel at times in moments of fatigue; but the essential clinical difference is that, while the mental effects of fatigue may be overcome by a sound night's sleep or a few days of change, rest, or diversion, the case of dementia is unaffected by these lighter remedial measures. In types of greater severity there is a tendency to automatic trains of thought which cannot be interrupted by an effort of the will or by ordinary diversity in the affairs of life. In the gravest types the whole delicate psychical mechanism of the mind becomes disarranged, and this manifests itself in the emotions, the will-power, and the capacity for thought. The perceptions, however, in this form of dementia do not suffer in anything like the proportion of the other faculties of the mind. This may at first sight seem singular, but, after all, it is only in the line of what we know of the physiology of the brain; for instance, the experiments of Münk, the German cerebral physiologist, have demonstrated again and again that the exsection of the auditory centre from the cortex of a dog does not impair at all the animal's capacity for hearing, but does away entirely with his capacity to understand the sounds that he hears clearly, as is evident by the motion of the head and the pricking of the ears. (See page 40.) After all, this is nothing more than a singularly

sharp demonstration of what, in a less definite way, has been known to generations of comparative anatomists—namely, that the cortex of the brain is the organ of the mind, and that it takes what we call mental cognizance of all the impressions coming to it from without.

The dementia resulting from mechanical or psychical shock is of sudden onset and is severe in its manifestations, so that the patient's memory, thought, expression, emotions, and will-power are often as completely blotted out of existence as by excision of the whole gray matter of the brain. Even these cases, however, if closely observed, will be seen to be receptive of sensations in a certain dull and apathetic manner.

Besides these dementias from cerebral exhaustion and mechanical and psychical shock, there are certain well-recognized types which almost all the writers upon mental affections have been able to define, and these are worthy of separate description. They are:

1. Primary, or acute dementia;
2. Hebephrenia;
3. Senile dementia;
4. Paretic dementia.

Paretic dementia is so thoroughly well marked as a form, is so well understood, and its prognosis and treatment are so radically different, that I shall reserve a separate chapter for it; but the others can best be treated in this connection.

PRIMARY DEMENTIA.

Synonyms: Stuporous insanity. Amentia. Primary stupidity.

Definition. A simple dementia occurring in young adults.

Clinical History. This occurs in young persons, as a rule under twenty-five years of age. It is characterized by a high degree of stupidity, so that the patient does not speak, makes very little volitional effort, cannot be interested in anything, and the emotions are either absent or are manifested erratically. These individuals sit quietly and stupidly, and when they have convalesced it will be found that they have little or no memory of what has taken place. The bodily functions are usually depressed, the pulse is slow, the appetite is either lessened or not manifested at all, although food will be taken often in the same quantities as in health if persuasion be used, the action of the heart is greatly enfeebled, the temperature is slightly lowered, the skin is cold, and œdema of the lower extremities is not infrequently observed. Ptyalism is a frequent symptom.

Causation. The causes of primary dementia are:

1. Heredity;
2. Masturbation;
3. Venereal excesses;
4. Anæmia;
5. Overexertion of mind.

Of all these causes, the only one that needs to be dilated upon is the last—that of mental overexertion. This, in my experience, has been the most frequent etiological factor, and, singularly enough, the

majority of cases have been in the male sex, although I am not certain whether or not this is a coincidence. The mental overexertion that I have seen has been that of overstudy, either actual or relative. Ambitious boys and girls are set to work to accomplish tasks that are frequently beyond their power; or even when their mental reach might be sufficient, the regulation of the habits is so faulty as to disable them prematurely by lack of proper fresh air, exercise, diversion, and regular and systematic habits of study. In some cases anæmia has also been an aggravating factor, but it is a curious fact that this has been the exception to the rule, and that while anæmia is rich in its pathological effects in other directions, it seems to be peculiarly infrequent as a cause of this mental type; indeed, most of the patients of this class are, so far as regards other than the cerebral tissues, in good health.

Prognosis. The prognosis of primary dementia is usually a good one, as most of the cases recover in from one to three months, and some may even terminate more quickly. In the infrequent cases that do not recover, the primary exhaustion has either been very severe, or there is improper treatment. Unfavorable cases pass into a condition of prolonged dementia.

HEBEPHRENIA.

Hebephrenia, or insanity of pubescence, is a mental disease observed more particularly in the male sex, characterized by a mawkish silliness with a tinge of melancholia. These cases are apt to be confounded with hysteria, and they stand upon the border-line in many instances between insanity and neurasthenia, although sometimes they may become so well marked as to constitute a genuine insanity. They are actuated by silly semi-delusions, a mawkish sentimentality, caprice, sadness that is not a true melancholia, or silly outbursts of hilarity that are temporary. In the lighter cases the impairment of reasoning power, judgment, and perception is slight, but in the graver ones these faculties may be markedly impaired.

Hebephrenia is easily distinguished from mania, melancholia, insanity, or primary dementia. It has not the true, continuous, intelligent sadness of melancholia, nor the post-cervical ache or insomnia; nor the continuous flow of ideas and hallucinations and hilarious delusions of mania, nor the restlessness; nor the continuous sedate silliness constituting true dementia; nor the period of continuous hilarity and good-natured flow of ideas and accentuation of all the physical and mental faculties of the maniacal stage in circular insanity, nor the passage from this mania into the continuous melancholy or depressed condition which is the second phase of circular insanity.

The prognosis of hebephrenia is good in the slight cases, but unfavorable in the severe ones in the fact that it is apt to be very protracted.

SENILE DEMENTIA.

At the outset it must be distinctly understood that senile dementia is something far above mere senility, being a mental condition that is clinically demonstrative of well-recognized pathological lesions. The mental picture is distinctly one of dementia or stupidity, and it is not precise to classify under this heading those cases of melancholia which occur in the aged, the more especially as they have an entirely different prognosis. The mind in its entirety suffers, presenting in one patient certain symptoms of the mental impairment, in another another, and in a third a different group; but however great the variations may be from case to case, through them all runs, to an expert eye, the unmistakable element of the dementia or stupidity. It may be slight or evanescent; but it is there if the history can be fully obtained. The judgment, the memory, the emotions, the sensations, one or more or all become affected; but the clinical demonstration of the impairment may be a shifting one. To these mental symptoms may be added paralysis in varying forms. Cases of senile dementia may therefore be divided into two great groups, namely: Mental symptoms alone, and those which have an admixture of mental and physical symptoms.

In the first group the memory is peculiarly apt to be affected. A sharp distinction, however, must be made between the impairment of memory which occurs in an aged person and the abnormal loss of it in one who is afflicted with senile dementia. The characteristic of the normal senile memory is that it becomes weak for recent events, whilst it may remain unimpaired for those of the past. In senile dementia, however, this loss of memory of recent events becomes much more marked, and in some cases, although not always, it varies from time to time. Thus, a senile dement may fail to recognize a person or a place that ought to be perfectly familiar to him, or he may do this at certain times and yet have a perfect memory regarding the matter at others. For this reason patients of this class are very apt to lose themselves upon the street, and I have known them to wander hopelessly for hours, walking and riding in horse-cars, in a city in which they had passed their lives; or to stare fixedly without a sign of recognition at the face of some near relative. The judgment becomes affected also in the majority of cases, so that a man or woman of hitherto clear and appreciative mind either utterly fails to reach conclusions or attains to entirely false ones. The emotions show a great variability, so that at one time without cause the patient weeps, or is morose, or breaks out into violent fits of anger, or becomes melancholy. The conduct grows to be erratic, and eccentric acts are performed that are either without reason or that are done for trifling motives, or the patient may have different degrees of restlessness, or even wander away from home. Delusions are frequent, and they may be either shifting or fixed. They may be based upon hallucinations or illusions, or arise without these. The delusions are apt to be of the nature of persecution, the patient imagining that he is being robbed or cheated, or that there

is some conspiracy against him, or that his relatives are treating him badly. Hallucinations and delusions are extremely frequent, most often optical or auditory in their nature, less frequently olfactory, and often based upon false perceptions through the general senses or through the sensations coming from the thoracic or abdominal viscera. Like all the other phenomena of senile dementia, the hallucinations and delusions are apt to be fragmentary and shifting in character, varying thus from time to time, although there are certain ones that are fixed, and in some rare cases all are of this permanent nature. Their variability may be at times almost startling, and their significance may escape one who is not expert in these matters. Thus, one patient of mine was taking a trip to Cuba, and whilst talking with the captain of the steamer, some three hundred miles from land, he suddenly said, "Look! look!" The captain looked in the direction he pointed, and saw nothing, and told him so. He exclaimed excitedly, "Oh yes, don't you see John," his coachman, "out there with the carriage and horses?" The captain got his glass and carefully scanned the horizon, but could see nothing, and yet the patient persisted for an hour or more that he had seen his coachman and the horses and the carriage three hundred miles out on the Atlantic Ocean. To my mind no more definite evidence of an hallucination could be obtained, and yet an expert was found who was willing to state that this might have been a mere mistake! It is often extremely difficult to make the distinction in these cases between an hallucination and an illusion, for the reason that it may be difficult to obtain the proof as to whether a given faulty perception is really at the start a faulty perception or is the transmutation of a correct perception into an imperfect one. Nevertheless this distinction can be made at times with great positiveness. For example, a certain gentleman about whose will there was a contest, had complained vehemently that there was a bad smell in one of his closets. The plumbers were summoned and the plumbing-work was removed, yet he persisted still in his belief that the disagreeable odor was there. The plumbers were again summoned, tore up the closet and were unable to find any pipes leaking or any dead animal. Still the patient insisted upon it that he noticed the disagreeable odor. So a sanitary expert was called in from the local Board of Health, but he could neither find any odor nor cause for any. The patient finally pasted up the closet, in a painfully laborious way with ordinary newspaper, which he plastered on with wet flour; and having done this, he declared that the smell had disappeared. His naso-pharyngeal passages were carefully examined at about this time by a competent laryngologist, and no cause for the odor was found in them. It was, however, testified to that some gas pipes had been leaking in the street at about the time when the old gentleman began to complain of the odor in the closet; so that the evidence was about as complete as it could be that a true perception, namely, of gas in the street, had been pathologically transmuted into a disagreeable odor in this closet, and no better evidence of an illusion can be given. These patients are also apt to become extremely erotic, and this condition although usually variable in its duration,

may at times become permanent, and sometimes even leads to the most brutal attacks by old men upon women or girls.

In the second group of cases of senile dementia, in which the mental symptoms just described are conjoined with paralytic phenomena, the paralysis is either of the nature of hemiplegia, monoplegia, implication of individual muscular groups, or, less frequently, individual nerves. When hemiplegia occurs it is of the ordinary type, causing motor paralysis of the upper and lower extremities and the lower part of the face, so that the naso-labial fold is weakened or lost on one side, and of the tongue, causing this organ to deviate to one side or the other. With this hemiplegia may be conjoined aphasia, usually of the ataxic form. When the paralysis is monoplegic, the upper or the lower extremity alone may be affected, or one side of the face. When individual muscles or muscular groups are implicated, some of the facial muscles or those of the upper extremity are most apt to suffer, although any of the muscles of the body may become affected. Individual nerves are seldom implicated, except the olfactory, the auditory, and the optic. In examining the olfactory it must be borne in mind that the sense of smell is often blunted in the aged, and that it can also be obtunded by naso-pharyngeal lesions; but an absolute anosmia that is not due to any naso-pharyngeal cause may be regarded as pathological. The mere blunting of the sense of sight, too, is a very general phenomenon of senility, but neuro-retinitis, hemianopsia, or amaurosis is pathological. It is very difficult to examine the sense of taste in a case of suspected senile dementia; but if it is possible to do this, it will sometimes be found to be impaired (page 161). With the exception of these cranial nerves, the paralytic symptoms of senile dementia are mainly motor in character, although, as has been said, it is difficult to reach conclusions about sensations in patients whose minds are affected. Tremor is another physical symptom that is extremely common, and it is situated in the tongue, the facial muscles, the head and neck, or the extremities, most usually in the tongue and the facial muscles. If this tremor is at all marked, it will render the handwriting tremulous and uncertain. Tremor, however, is so general a symptom in the aged that, taken by itself, it is of little value diagnostically, but, becomes of enormous importance when it is conjoined with the other symptoms of this disease. The speech is affected in various ways, according to the cause of the derangement. Thus, if the patient be deaf, it may be low and mumbling or high-pitched; or it may have the characteristics of the various forms of aphasia (page 141); or if the tremor is great, it may be muffled, low-pitched, and monotonous.

Prognosis. Senile dementia is usually of very unfavorable prognosis, although quite a number of cases can be treated with such success as to make the ordinary changes of senility again become almost normal.

For the purposes of the general practitioner but little time may be spent upon the forms of secondary dementia, for reasons which will

appear. It occurs either during the convalescence from the acute or subacute forms of insanity, or in the terminal stage of the organic mental affections, or with grave textural lesions of the cerebrum.

DEMENTIA, OR MENTAL EXHAUSTION IN THE CONVALESCENCE FROM CURABLE INSANITY.

This form ought not really to be called dementia, not because the phenomena are not those of stupidity, but rather for the reason that the term is apt to be misleading. They are really conditions of cerebral exhaustion after an acute or subacute insanity has run its course, and they mark the beginning of convalescence. I simply dwell upon them because I have so often seen physicians and households thrown into despair by their supervention, when a more accurate knowledge would have seen every reason for hope. It is not every form of curable insanity that passes through this stage, curiously enough; but it does occur at times, and, as will be seen in the section upon treatment, it calls for no special remedies other than what are indicated by the disease which has preceded it.

TERMINAL DEMENTIA.

Very different, however, is the form of dementia which occurs with certain organic diseases of the cerebrum in which insanity has been one of the symptoms, as in paralytic dementia, or which occurs toward the end of a case of brain tumor, cerebral hemorrhage, embolism, or thrombosis, or after a cerebro-spinal meningitis, or a cerebral abscess. This type marks the final stage, and unless its cause can be removed—which is very seldom the case—the prognosis is hopeless.

Pathological Anatomy of Dementia. The pathological alterations of dementia are unknown in all the forms except the senile one. In this the lesions are those of precocious senility, so to speak, consisting of the degenerated vessels, meningitis of the pia, occasionally of the dura mater, and the textural alterations of the cerebrum resulting from these arterial and membranous changes. Atheroma of the vessels is extremely frequent, as are also miliary aneurisms. Arterial thrombosis may occur, and also embolism, of either intracranial arterial origin or from the heart. Intracranial hemorrhage frequently occurs, being either the result of rupture of one of the larger vessels in which atheroma is especially prone to occur, or from miliary aneurism of the arterioles of the basal ganglia or the cortex. Softening of the brain is frequent, too, induced by the arterial changes resulting in atheroma, miliary aneurism, thrombosis, and embolism. It is a question as yet undecided as to whether cerebral softening ever occurs without these precedent arterial changes. Meningitis of the pia mater and the dura is not infrequent. As has already been said (see chapter on "Paralytic Dementia"), Tuczek

has described loss of the tan gential nerve-fibres in certain cases. That alterations in the ganglion-cells of the cortex occur would seem extremely probable, but the nature of them is not as yet thoroughly understood. When the ordinary senile changes in the peripheral vascular system and the heart are, as is usually the case, superadded to these pathological alterations of the cerebrum, it is easy to understand the mechanism of production of the varied symptoms, both physical and mental, of senile dementia. The whole machinery of the brain is disarranged, and the symptoms of the disease are, therefore, as we have seen, capable of ranging over as wide a field as is covered by the functions of the senile cerebrum. The mental symptoms are probably due to the fact that the frontal lobes are especially affected. The paralytic symptoms arise from lesions of the motor area (page 139) or the internal capsule (page 142). That some of these paralytic symptoms are permanent, whilst others are temporary, may be dependent upon two factors, namely, the localization of the lesion and the nature of it. For example, the arterioles of the pia mater ramifying over the cortex anastomose freely with one another, as we have seen (page 92), so that hemorrhage, thrombosis, or embolism in one of them may at first produce considerable impairment of function, which may disappear in a short time, as anastomosing vessels bring in an additional supply of blood to the affected part. On the other hand, when a terminal arteriole of the internal capsule is diseased, the area of territory supplied by it has no anastomosing vessel to feed it when its blood-supply is cut off by an embolus or thrombus, so that the injury primarily inflicted by such embolus or thrombus remains unmodified. Then, too, the extent of the hemorrhage and the rapidity of its supervention, the extent to which an embolus or a thrombus blocks up a vessel, and the rapidity with which it does so, are all factors determining the clinical symptoms of the lesion. The alterations in the cerebral lymphatic system have been but little studied, but they are probably of a similar nature to those in paralytic dementia. (See chapter on "Paralytic Dementia.")

Diagnosis. The diagnosis of the different forms of dementia is usually not difficult. The element of dementia or stupidity is the salient factor, and it is usually very easy to obtain the history of preceding organic disease of the cerebrum or lack of it, so as to distinguish the primary from the secondary or terminal dementias. The differentiation is to be made from the following affections:

Melancholia;
Katatonia;
Delirium;
Paralytic dementia;
Bromism;
Morphinism;
Cocainism;
Alcoholism;
Apoplexy;
Confusional insanity;
Organic disease of the cerebrum;

Nephritis;
Diabetes.

The essence of melancholia is, as the name denotes, melancholy, whilst that of dementia, as is equally well indicated by the name, is stupidity. Herein lies the fundamental difference between the two diseases. In certain grave cases of melancholia, as in the attonita form, or in that occurring in the senile, there is such a degree of taciturnity as almost at times to be mutism, and to a superficial observer this may seem like dementia; but careful observation will elicit evidences of memory and perception and reasoning power but slightly impaired. It is a very common mistake to call melancholia occurring in the aged senile dementia. Practically, the difference is great, because the prognosis and the treatment of the two diseases are entirely different, and so are the medico-legal bearings. Moreover, the senile melancholic often has a very pronounced suicidal tendency which is seldom present in the senile dement.

The succession of symptoms in katatonia covers a range that is never seen in any of the forms of dementia, and this alone should make the diagnosis an easy one.

In delirium there is a dream-like condition with hallucinations and illusions, and usually a history of preceding febrile disturbance, and the temperature is generally increased during a certain course of the mental manifestations.

Paralytic dementia may be difficult at times to distinguish from senile dementia, but the former disease occurs in much younger adults, rarely, indeed, in the aged; the paralytic attacks are more of isolated muscles and muscular groups rather than hemiplegia, and its delusion of grandeur, which is frequent, is never observed in senile dementia.

Bromism may produce very great stupidity, and has often led to a diagnosis of dementia. The distinction, however, is easily made by the history of the bromides having been given in large doses, by the disagreeable breath, the dilated pupils, and the loss of the pharyngeal reflex that is normally observed in tickling the throat with a spoon or a feather.

Morphinism will sometimes induce conditions simulating dementia when overdoses have been given or when the drug has been suddenly withdrawn. In such instances the history will enable one to make the diagnosis. Where this is absolutely lacking, as in ambulance cases, the contraction of the pupil may throw light upon the matter, when it is markedly contracted. When the pupil, however, is widely dilated, as in certain cases of morphinism under the circumstances described, the diagnosis may become a difficult one, and time alone can make it. Indeed, it is not always safe to rely upon the contraction of the pupil, for this may be due to lesions within the eye itself. When there is any doubt about the matter, sharp interrogation of the patient will often bring out the truth, as very few morphine-fiends have any hesitation about confessing to their vice rather than suffer the torment of abstinence.

In a suspected case of cocainism the sensibility of the nostrils

and the throat should be tested, as they may retain a certain degree of anæsthesia if the cocaine has been taken through them. If no information can be obtained in this way, or through the history of the case, it will not be safe to attempt a diagnosis until the effect of several doses of cocaine has been tried. If this does not immediately disperse the stupidity, it can be definitely said that the dementia is not due to cocainism.

In a case of dementia from alcoholism the history is very important. If this is lacking, the urine should be tested for alcohol, and the condition of the facial capillaries should be carefully observed. If they are dilated in the usual way, and there is the alcoholic odor of the breath, the diagnosis is easy. In some cases of alcoholism, however, there may be great facial pallor, and in such an event the odor of the breath may be the only factor for diagnosis. If no data can be obtained from the history, breath, dilatation of the facial capillaries, or alcoholic urine, the diagnosis should be held in abeyance for the time being.

By apoplexy in this sense are meant the phenomena which are included in the chapter upon "Hemorrhage, Embolism, and Thrombosis." In dementia suspected to be from these causes the evidences of paralysis should be carefully sought for. It should be observed whether the cheeks blow out symmetrically on the two sides in expiration; or if the respiration is not sufficient to expand the cheeks, the patient should be pinched or pricked with a pin, and during the resulting cry it should be carefully observed whether the facial muscles contract equally upon the two sides. The attitude of each upper and lower extremity should be carefully studied so as to see whether there is more muscular flaccidity than in the corresponding member or members of the opposite side; and the foot and the hands should be pricked with a pin upon the two sides to see whether the resulting movement is equally free in all. The knee-jerk should be carefully tested upon the two sides, and it will often be found to be lost or exaggerated upon the paralytic side. If it is possible to make the patient protrude the tongue, note should be taken as to whether this protrudes straight or deviates to one side.

In functional insanity the element of dementia is manifested rather in the acts than in the conversation, and there is never such a degree of stupidity as obtains in primary dementia, or such a degree of agitation as is seen in hebephrenia.

In all cases of dementia organic diseases of the cerebrum should be carefully excluded. To this end it should be ascertained that there has been no symptom of abscess, either idiopathic, proceeding from middle or internal ear trouble, or from venous thrombosis; that there is no paralysis of motion or sensation; that there has been no injury to the skull; or that there has been no symptom of intracranial tumor or meningitis.

The diagnosis from nephritis is easily made, because a nephritis that is sufficient to produce a mental condition simulating dementia will show large amounts of albumin and the different varieties of casts in the urine. It must be remembered, however, that small

amounts of albumin and a few hyaline casts, even a few epithelial ones, are not sufficient for a diagnosis of nephritis as a cause of dementia. The crucial test in this particular will usually be the estimation of the urea, for if this is far below the normal the dementia may be set down as of nephritic origin.

In diabetes the diagnosis is also made with ease, as in a case of dementia from this cause there would be found a high specific gravity of the urine and large amounts of sugar.

TREATMENT. The treatment of the dementias will vary, necessarily, according to the type. It may at once be said that the terminal dementias are not treatable; that is, those which indicate the terminal stage of organic disease of the cerebrum.

The primary dementias resulting from exhaustion of the brain, emotional disturbance or mechanical shock, should be treated by absolute isolation and such measures as may be indicated by the general nutrition of the patient and the circulation. If the patient has been in good health, nothing more may be needed than complete isolation and rest. This should be carried out by putting the patient to bed, and excluding all but those in immediate attendance upon him or her. Where it is possible to do so, it is always best to have a trained nurse in these cases, as in this way old associations are absolutely excluded; and besides this the neatness, the noiselessness, the tact, the professional nonchalance and coolness of the trained nurse can never be attained by an anxious member of the family. If this isolation in charge of a trained nurse can be carried out away from home, it is almost always productive of better results. The robust patient may need little more than this isolation and care, continued for a few days, perhaps a week or two. At the end of this time short walks or drives, or some gentle means of diverting the mind, such as some form of light reading, may be mentioned. It is the greatest folly in the world, however, always productive of bad effects, to attempt to divert these cases by relatively energetic amusements, such as going to the theatre, travelling, or matters of that sort. They will do far better always with the absolute mental and physical rest. As time progresses, other gentle means of diversion should be devised, always taking care, however, to err on the side of too much rest rather than too little, watching carefully the effect of each new outlay of energy, and not repeating it if the patient becomes restless or more listless.

Should the patient, however, be anæmic or exhausted, or in a poor physical condition, with poor appetite and defective circulation, much more energetic and elaborate measures should be taken. The treatment then should become the same as in neurasthenia, and the reader should carefully study what has been said on pages 463 et seq. There may be, however, a slight difference needed in the treatment, in the fact that these cases of dementia will do best if a cardiac stimulant is employed early in the treatment, and sometimes it may be advisable to use it from the very start. If the stupidity is very profound, and the circulation is poor, an alcoholic stimulant should be resorted to, varying according to the needs of the case from an ounce

and a half to three ounces of whiskey in divided doses in the twenty-four hours. It is often surprising to see how well these patients will bear this large amount of alcohol; and in some instances I have even given with benefit as much as 8 to 10 ounces in twenty-four hours, although this is scarcely ever necessary except in those who have been accustomed to the use of alcohol. If the alcoholic stimulant acts well, a special cardiac stimulant will not be necessary. In some instances, however, especially in those who have been extremely temperate, alcohol will cause considerable restlessness without any coincident improvement in the mental symptoms; and when this is the case it should be either discontinued entirely or reduced to a small quantity, and the sulphate of strychnine administered in its place. The dose of this should be from $\frac{1}{50}$ to $\frac{1}{30}$ grain three times a day, best in the form of tablet triturates of a reliable firm, as in them the dose is exact, whilst in a solution it may vary with the evaporation of the fluid or from the carelessness of the person giving it. Some patients cannot take strychnine because of the disagreeable griping produced by it in the unstriped muscular fibres of the intestine, and when this occurs, digitalis should be substituted, 10 drops three times daily of a freshly made and reliable tincture, or one grain of the solid extract in pill-form at the same intervals, or three drops of a reliable fluid extract. When the patient has been restored to such mental health as to make it seem as if he or she were in the usual condition of mentality, a vacation of two or three months should always follow, as it too often happens that return to the ordinary course and associations of life at this period will bring about a relapse. It is at this time that travel becomes of great value if undertaken with due regard to comfort and avoidance of fatigue.

In primary dementia the same treatment should be applied.

In hebephrenia it is not always necessary, however, to advise rest to the same extent, although isolation should be carefully maintained. It is extremely important in cases of hebephrenia to take the patient away from home and surroundings and put him or her in charge of a trained nurse, because many of these cases are those of young adults who have never had any self-control and who have never been taught it by parents or relatives; furthermore, these individuals will always do better with strangers. In some of them the agitation and restlessness are so great that they may call for the administration of a cerebral sedative, and the best of these are the bromide of potash and the hydrobromate of hyoscine. The former should be given in doses of 20 to 30 grains *pro re nata*, and the latter in the same way in doses of gr. $\frac{1}{100}$. Usually 20 grs. of bromide of potash once or twice a day or $\frac{1}{100}$ gr. of the hydrobromate of hyoscine as often, will be sufficient; or, if either dose alone does not quiet the patient sufficiently, it may be well to combine the two drugs in the above dosage. These cerebral sedatives, however, should not be used except when necessary to restrain the restlessness that is sufficient to weaken the patient, for any less degree of agitation will be best overcome by treatment of the disease itself. Hyoscyamine should never be used in these cases, for it is a dangerously depressing drug

in many instances, and the disagreeable dryness of the mouth and dilatation of the pupil which it produces are apt to increase the dementia.

Senile dementia should be treated upon the same principles as the primary type. In many instances, however, the rest should be more absolute.

There is no necessity of sending these cases of dementia to an asylum, for not only can nothing more be done for them there than at home, but generally far less is attempted, and in the curable cases the associations of an asylum are extremely disadvantageous when the stage of convalescence has been reached, whilst the stain of having been in such an institution may constitute a serious obstacle in their after-life.

CHAPTER XVI.

CONFUSIONAL INSANITY.

Definition. Confusional insanity consists of a slight stupidity that is characterized by incoherence and confusion of ideas rather than by true dementia, without emotional disturbance.

It is often confounded with true dementia, but the difference is that the dement is positively stupid, whilst in this form of mental disease there is a stupidity that is inherent rather to the ideas and the actions than to the general appearance. For example, a woman afflicted with this form became widely known several years ago by her action in kidnapping the child of well-known people. She was finally found living at a hotel in a neighboring city with the girl whom she had abducted. She could see nothing wrong in what she did, was very much surprised that anybody had found fault with her, claimed that she had taken the child only because she took a fancy to it, had treated it very kindly, had not attempted in any way to conceal herself, and had made no attempt whatever to obtain any pecuniary reward. After a long conversation with her, during which I elicited these facts, she started to leave the room, and calmly lifted a Bible as she passed by a table, and was walking off with it, when I stopped her and called her attention to the fact that the book did not belong to her. She made no attempt to conceal her intention to take it, but placidly remarked that people took her things, and she did not see why she could not have theirs. Conversation with her upon general topics would not have evidenced any mental aberration except to one skilled in the examination of deranged minds. This woman was a type of this form of insanity. In some cases the memory is blurred, but usually it remains intact. Rarely there are hallucinations and delusions, both being usually varied and non-characteristic, except that delusions of identity are common. The speech is sometimes confused, although this has been the exception in the cases I have seen, and there is no tendency to extravagance, but rather to irrelevancy and incoherence.

The prognosis of these cases is usually good, although, when the attacks are frequent or when the patient is much debilitated, the disease may become chronic.

The diagnosis is readily made, and the affection is to be distinguished from dementia, mania, general paresis, melancholia, and insanity of pubescence. (See Part III., Chapter III.)

The treatment is to be along the same line as in dementia. (See Chapter XV.)

CHAPTER XVII.

PARANOIA.

Synonyms: Verrücktheit. Primäre Verrücktheit. Chronic delusional insanity. Délirants chroniques. Monomania. Reasoning mania.

Definition. Paranoia is a form of mental disease characterized by logical or systematized delusions of persecution and self-exaltation, without excitement of emotion or idea or impairment of memory.

History. The word paranoia is derived from the Greek word *παρανοία*, which was a classical term denoting insanity used by some of the best Greek authors, such as Æschylus, Arrianus, Plato, Aristotle, Lucianus, and Plutarch; but it does not seem to have been used by any modern writers until it was employod by Vogel in 1764 as a collective name for some nine different forms of mental disease. In 1818 Heinroth also made use of it to denote certain secondary states of exaltation with fixed delusions and exaggerated feeling of personality, although the first use of the word in its modern meaning is due to Mendel. The evolution of our present knowledge of this form of insanity is due primarily to the observation of the French alienists of fifty years ago, then to that of the Germans, whilst in recent years the Italians have added very considerable material, and at present the French are again interesting themselves in the subject. Singularly enough, in this scientific discussion that has been one of the most interesting and critical of modern psychiatry, the English and the Americans have borne but a small part, with one or two notable exceptions. The germ of paranoia was first detected by the great Esquirol in his chapters upon monomania, with ideas of grandeur. Griesinger in 1845 next described the combined or successive delusions of persecution and grandeur. In 1852 Morel detected what he termed the *systematization*, or, in more Anglo-Saxon terms, the logicality of the delusions. With this the picture of the disease was drawn in its essentials, so that the discussion from the days of Morel to the present time has waged around the important but relatively subsidiary queries as to whether the malady is hereditary and chronic or secondary to pre-existent insanities, or whether it is primary and can occur acutely and non-hereditarily. Griesinger, in his first description, in 1845, was of the opinion that it was always a disease secondary to mania and melancholia, but twenty-two years afterward he admitted that Snell was correct in his statement that it was always a primary disease. Snell, however, himself described in 1873 a form secondary to melancholia, mania, and epilepsy, and at the present time most of the French and Italian authors are agreed with him in

the belief that there are both a primary and a secondary variety. The question of heredity has undergone quite as revolutionary a transformation. The earlier authors were very positively of the opinion that paranoia was always a hereditary disease in the sense that it occurred in those who were predisposed to insanity or to neuroses by heredity, and even at the present day some of the best-known alienists are still of this way of thinking; but in 1878, in his celebrated Hamburg discourse, Westphal described an acute form and initiated a literature upon that point, in which his views have been espoused by Leidesdorf, Koch, Jung, Schüle, Merklin, Morcelle, Marie, Tonnini, Buccola, and others, so that some recent authors, especially Werner, have gone so far as to include under the heading of acute paranoia many cases of puerperal, post-febrile, alcoholic, morphinic, and cocainic insanity, and the semi-insane neuroses, such as *folie de doute*, fear of certain localities (agoraphobia, claustrophobia, topophobia), dipsomania, pyromania, kleptomania, certain homicidal and suicidal impulses, certain belonephobias, certain morbid impulses to collect pins, onomatromania, certain morbid impulses to gather figures (arithmomania), and finally an almost laughable form described by Magnan in certain old maids who have the characteristic old-maid love for cats, and to which he gives the magniloquent name of *folie des anti-vivisectionistes.* In France of late years Magnan has taken some peculiar views of the subject, which have met with but partial acceptance even in his own country. He claims that the chronic delusional insane have been described in two phases of the evolution of their mental disease, and that to these two phases has been given the name of paranoia, so that systematized delusions of persecution and grandeur are but stages in chonic delusional insanity; but as the terminal stage of the cases he describes is dementia, and as very few cases of true paranoia pass into dementia, it is obvious that his description does not tally with the facts in this particular, whilst the acute cases of paranoia usually recover. From this review it will be apparent that there is a mental disease characterized by delusions of persecution and self-exaltation, and that this disease may be hereditary or non-hereditary, chronic or acute, primary or secondary.

Clinical History. Paranoia may be divided into, first, the primary, and secondly, the secondary form.

The primary form may be acute or chronic, and both the acute and the chronic may be hallucinatory.

Acute primary paranoia begins with a delusion of persecution which may be infinite in its variety. At first this harasses and vexes the patient, and may even cause doubt in his own mind as to the genuineness of the delusive phenomena; but as time passes he becomes more and more convinced of the reality of the delusions, and it becomes correspondingly difficult to reason with him about them. He lives in an atmosphere of suspicion—everything around him has a covert meaning, and "trifles light as air become as proof of Holy Writ." Indeed, Shakespeare's description of Othello's jealousy is an excellent picture of what is often seen in alcoholic paranoiacs. People are watching him, he thinks, on the street, and in the

public conveyances; somebody coughs as he passes by, and immediately he believes that that cough has some secret meaning; a match lies before his feet as he goes along the street, and he checks himself and goes around it, convinced that there is some design about it; his wife fastens a handkerchief in the window to dry, and he rushes headlong to the conclusion that this is a signal set for some paramour; or a headline in the paper, a bill-poster on the street, a sign over a store, a casual glance from a passer-by, an advertisement, may have a fantastic meaning lurking in it to his distorted perceptions. As time passes these delusions become more and more fixed. The patients reason about them calmly, perhaps, and they differ from the delusions of melancholia and mania in the fact that any agitation in the patient is produced as the consequence of these delusions, and the excitement does not precede the delusions. The delusions are thus always bound together in a chain of false logicality, in which, however, the morbid element is plain to the experienced observer. As the delusions become more and more fixed, there gradually arises a question in the morbidly logical mind of the patient as to why he should be thus persecuted, and out of this, in a natural enough way, gradually grows the delusion of self-exaltation, or what the French somewhat magnificently term the delusion of grandeur. The patient then becomes in his own estimation a person of importance, either because of his station in the world, or because of his personal attributes. Thus, Dougherty, the would-be lover of a well-known actress, who believed that he had been persecuted and wronged because of his love for her, stated to me calmly and with a half-deprecatory smile that he had been the handsomest man in the world before he had been broken up by confinement and anxiety. Another patient told me in a smiling, modest way that he was persecuted because he was possessed of a certain secret way of polishing wood, in which his skill was greater than that of any other living man. Another believes that he is watched, and that his food is poisoned because he has certain business secrets, the disclosure of which would be very damaging to Jay Gould, the great American arch-millionaire, the monetary anti-Christ to hordes of diseased minds and many sane ones. Another imagines that he is a great man in the estimation of the world because he has made great scientific discoveries, which his rivals are trying to discover; and so on through a myriad phases, infinite as the mind of man in its various degrees of culture and imagination, these patients have many delusions of self-importance. The judgment, the reasoning power, and the memory of these paranoiacs are remarkable, indeed, often startling, so that they are almost certain to impress the lay observer and many physicians also as being perfectly sane except upon certain points. One uneducated man outlined to me the most remarkable theory of the action of the mind upon the muscles, embodying in all its essentials the views of the most advanced neurologists of the present time regarding the motor centres of the cerebrum; whilst a woman of this class was brought to me by her lawyer, who was a perfect believer in her sanity, and who told me the most connected story of the wrongs that

she had suffered at the hands of relatives, all of which were proved to be entirely false in the light of subsequent developments; and a little Methodist minister once came under my care who calmly narrated to me the most circumstantial account of proofs of his wife's infidelity, so connected and so circumstantial as to stagger me for the time being, the more especially as he maintained them with great distress of mind when brought face to face with his wife, and when I told him frankly that he must be either perfectly sane or else have a delusion and be insane, he promptly and calmly admitted the force of the argument, and was entirely willing to wait for the test of time, which, however, showed incontestably that he was insane. I have seen a patient of this class take the witness-stand to demonstrate his own sanity, answer questions with perfect readiness and logical sequence, stand unscathed an analytical cross-examination, stagger judge and jury, afterward point out the logical errors of the trial, and yet in the next breath say something that would evidence a firmly rooted delusion. These patients write letters coherently and well; indeed, often undertake literary work; and at one time in the Utica asylum in New York State a magazine was conducted with considerable skill by a number of patients of this class. Yet, underneath all this seeming good judgment and logicality, the delusions and the false judgments based upon the delusions keep surging to the surface. The loss of the ethical sense, too, in these patients is remarkable, and this symptom is one upon which writers have not laid by any means the stress that its importance and frequence deserve. They will tell you of some brutal act that they have done with the utmost calmness and the most perfect apparent recognition of the nature and quality of the act and its consequences. Thus, this man Dougherty, to whom I have already alluded, told me very calmly that when he had been committed to the asylum twice, and found that he had no redress, he made up his mind that the system of committing people to asylums was defective; that he sought for a long time for the best means of correcting the evil, and that he came reluctantly to the conclusion that a revolution, as he expressed it, was the only effective remedy, so that he determined to kill the physicians who committed him, the judge who had indorsed the commitment, the commissioners of lunacy who had approved of it, the officers of the asylum to which he had been committed the second time, the local commissioners of charity in whose charge the asylum was, and the State commissioners of charity who had visitatorial power over all the asylums of the State. He armed himself with two revolvers and proceeded to the Flatbush Lunatic Asylum, where he shot a young assistant physician, Dr. Lloyd, backed out of the asylum, keeping everyone at bay with his two revolvers, and proceded on the horse-cars to the Brooklyn Bridge, a distance of some seven miles, to cross over to New York, where he had proposed to go on with his murderous work; but fortunately he was arrested on the bridge at the instance of another physician who had followed him. He said that he was very reluctant to kill Dr. Lloyd, because he knew that the latter was a young man whom his parents had educated at considerable

sacrifice, and because he was the only son; but he steeled his heart to the deed by reflecting upon the necessity of the revolution which he was thus to inaugurate. He also said that he had at first intended to shoot the cook of the institution, but second thought convinced him that people might think that there was an element of spite in this which would detract from the moral effect of the deed. I remarked at this point that I thought myself that it would have been rather a small affair to kill the cook, whereupon he quickly rejoined, with a chuckle, "Oh, if you had eaten the food that that d—d scoundrel cooked, you would have wanted to kill him, too!" and laughed heartily. Nothing could impress upon him the enormity of his deed or of the plan that he had in mind. This is a typical illustration of the cold-bloodedness, or, to speak more accurately, the loss of the ethical sense, which is a prominent symptom in these patients. Their delusions, indeed, approach the nearest of all the insane delusions to the fixed and erroneous beliefs of the sane, for they will reason about them logically up the last point of being convinced, and it may happen that in the earlier stages of the disease it may be possible to convince them for the time being of the falsity of their opinions. Between their delusions and the fixed beliefs of the sane, however, there lies this great difference, namely, that the latter do not have the predominant delusion of persecution mingled sooner or later with that of self-exaltation. In a scientific sense, therefore, these patients are not responsible. Even the dictum of Anglo-American jurisprudence, which is embodied in the statute of the State of New York declaring that a man shall be held responsible for his acts if he knows the nature and the quality of his act and the consequence thereof, does not make these patients responsible, because while they may, parrot-like, state that they know the nature and quality of the act, and what the punishment will be for it, it is very evident to the most superficial observer that they do not really appreciate what they are doing because of their lack of ethical sense and because of the manner in which the mind as a whole is warped by the fixed delusions of persecution and self-exaltation. It is quite another question, however, as to whether these patients should be punished by law. There can be no question that they are very dangerous lunatics, and they should certainly be put under restraint for such time as to make it certain that they are really cured.

These patients are generally subject also to paroxysms of excitement, sometimes arising from their delusions, sometimes entirely independent of the latter, and in these periods of excitement they are apt to become confused and incoherent, although such phases are usually temporary.

The chronic form of simple paranoia may either be preceded by the acute form, or it may be chronic and gradual from the very onset. Under this chronic form are to be found what Sander called original paranoia, litigious paranoia (*paranoia querulantium*), moral insanity, erotomania, and the inventorial and the sexual paranoia. The truth of the matter is that classification of this kind is an endless task, for the delusions of paranoiacs are as various as are the

different sensations, and the metamorphoses of these delusions with the hallucinations that occur in the classes of which we have yet to speak are simply chaotic. Nevertheless, the litigious, the moral, and the original forms deserve a more precise description because of their frequency.

Litigious paranoia is generally to be found in those who have a neurotic heredity. The onset is gradual, and it is usually caused by some loss of property or injury of reputation, out of which arise the delusions. These patients press from suit to suit without regard to whether they win or are defeated, and are never-ending nuisances, often dangerous ones, to juries and judges. A case of Buchner's formed an association of the oppressed; another began a crusade for the liberation of the supposed insane from the asylums; another, being temporarily committed to prison, organized an association of the murderers, whom he regarded as deprived of their rights as citizens, and cheerfully wrote to my friend, Dr. Hammond, that he, the patient, was now president of the Murderers' Club, and that false witnesses would hereafter be dealt with in a summary way that was much more in consonance with justice than the tardy processes of the courts.

Moral insanity or moral paranoia is invariably found among those who have a neurotic heredity. In these cases the moral defect may begin in early youth and continue throughout life. The acts of these individuals are usually without motive, although, when the passions or prejudices of people have been aroused, they may seem to be nothing more than the deeds of rogues; but the useless theft of small articles by a person who is abundantly able to pay for them, the obtaining of money upon false pretences by an outlay far greater than is represented by the money thus obtained, without estimating the loss of time and labor, the utter lack of appreciation of the consequences evidenced by the act, and very frequently its stupidity, are all very evident to the dispassionate or unprejudiced and experienced observer. I have known a patient of this class spend ten dollars to swindle a poor woman out of a dollar, or lose a position worth hundreds of dollars a month by obtaining a few dollars by false pretences, or cheat a person by false representations into buying a farm that he could have made infinitely more money by retaining. In all these cases of moral insanity that I have seen, however, there is, if not continuously, at least paroxysmally, the delusion of persecution and self-exaltation.

The original paranoia of Sander occurs in those who are hereditarily neurotic, or who present anatomical signs of degeneration, such as cranial deformities, and defects of development, as stuttering, left-handedness, total loss of muscular sense, absolute lack of any sense of humor, etc. This is an infrequent form. It generally begins at puberty, although the earlier youth may have presented vague and indefinite signs of eccentricity, such as a peculiar reserve, coldness of manner, suspicious attitude, visionariness, a vague something that impresses people with the idea that the patient is peculiar.

Acute hallucinatory paranoia is, as the name denotes, accompanied

by hallucinations, often illusions, which are at times extremely vivid and detailed with a picturesqueness that portrays the mental impressions they have made. This form occurs most frequently in the inanition succeeding acute febrile disorders, such as typhus and typhoid, in the puerperal condition, in peritonitis, after operations, and many cases have been seen after the recent epidemics of influenza. Alcoholism, morphinism, and cocainism are also not infrequent causes. This hallucinatory paranoia may also occur in a chronic form.

Secondary paranoia is seldom seen, if I may judge by my own experience, and when it occurs, it is generally after mania and melancholia, passing, however, either into convalescence, dementia, or a relapse of the original mania or melancholia. It has been claimed that it sometimes occurs after some of the other insanities, but this I have never seen.

These paranoiacs constitute the most dangerous class of the insane, especially in the acute and chronic simple form; and the deeds which they commit are the dangerous ones of a deluded patient who is yet possessed of all the intelligence that can make a deed dangerous. The history of the world is full of the brutal murders that have been committed by this class, and all the educational means of modern civilization, such as the newspapers, the magazines, and books, which go to educate and elevate the minds of the sane, have an equally powerful but perverted effect upon these warped intellects. They are as much the enemy of the sane as the wild beast is the enemy of man. Their delusions are so fixed and their intellects otherwise are so keen that there can be no truce between them and the sane. They live in a world of their own, and the sane appear to them as abnormal as they appear to the sane. Out of their delusion of persecution necessarily grows the necessity of action to protect themselves from the persecutors; the logic of events continually presses them on, however sluggish and cowardly they may naturally be; they are irritated and harassed and vexed and troubled into aggressiveness, and, like Mahomet, they enforce their opinions with the sword. In this respect they differ radically from the melancholiac, who passively suffers the injury that he believes is being done him, and which he attributes to some fault or sin of his own, and not, like the paranoiac, to the persecution of others; and their delusion of persecution is entirely different from that of the case of mania, who is simply terrified by it and proceeds to no logical or connected act, but only occasionally to an impulsive one.

Prognosis. The prognosis in the cases of acute simple and acute hallucinatory paranoia is generally good, for the disease usually terminates in recovery, infrequently in a chronic condition, and very seldom in dementia. In the chronic cases, however, the prognosis is very unfavorable, especially in those of a neurotic heredity and in the original form of Sander.

Etiology. The causes of paranoia are:

Heredity;

Traumata;

Mental and physical exhaustion;
The puerperal condition;
Febrile diseases, such as typhus and typhoid;
Surgical operations;
Epilepsy;
Alcoholism;
Morphinism;
Cocainism.

These causes need not be gone into in detail, as they are considered in the historical portion of this chapter or in other chapters.

DIAGNOSIS. The diagnosis of paranoia should be from—

Mania;
Melancholia;
General paresis;
Hallucinatory insanity.

In mania there is an acute or subacute onset, the delusions are but slightly systematized, whilst the emotional condition of hilarity is excessively well marked, and there is a larger degree of motor restlessness than is ever seen in paranoia.

In simple melancholia the delusions may sometimes be so logical as to puzzle us temporarily, especially when they take the nature of persecution; but in a very short time there will supervene the suicidal impulse, the confusion of mind, and the profoundly melancholiac condition. In the simple form of melancholia, too, the insomnia, the post-cervical ache, and the characteristic *facies*, of which I have elsewhere spoken, are symptoms not observed in paranoia.

In general paresis the dementia is a prominent feature throughout the disease, the delusions are always stupid and illogical (unsystematized); there are also the physical symptoms of pupillary inequality, abnormal pupillary reflexes, and tremor of the tongue, facial muscles, and limbs.

The great rush of ideation in hallucinatory insanity, with the utter lack of coherence in the sequence of ideas, is the direct reverse of the logical and emotionless delusions of the paranoiac, whilst the variations in the former disease from motor and mental excitement to depression or exhaustion and obstinate taciturnity are also a feature that is not observed in the latter disease.

TREATMENT. The treatment of paranoia should vary accordingly as it is acute or chronic. In the acute cases any causes of inanition should be removed at once; and in the post-febrile cases occurring after typhus and typhoid fever and influenza, nourishment, alcoholic stimulants, and rest are generally all that is needed, and narcotics should not be employed at all, or used very carefully. The feeding in these cases should be far in excess of what is normally taken by the patient, if it is possible to give this amount of food, and I am never satisfied unless I can have the patient take at least three full meals a day, with a pound of beef made into a strong beef-tea, and two quarts of milk. Potassium bromide, hydrochlorate or hydrobromate of hyoscine, opium, and hyoscyamine are the best drugs with which to control the delusions and hallucination in the cases that are

not due to marked inanition, and even in these they may be sparingly employed if large quantities of food and stimulation do not answer the purpose. The dose of bromide of potassium should never exceed 20 grains, three times a day. The hydrochlorate or hydrobromate of hyoscine in doses of $\frac{1}{100}$ grain once or twice, or even in robust individuals three times a day, is an exceedingly useful drug in controlling the restlessness and the delusions, and the great advantage of it is that it can, if necessary, be given for weeks at a time in hardy individuals. Hyoscyamine is an excellent drug in many cases for overcoming the motor excitement and the tendency to violence, and it is often useful in combating the hallucinations; but it is apt to be very depressing in its effect and should only be used temporarily, care being taken even then to see that it does not produce retention of urine or even strangury. Opium, in the form of the solid opium or as an aqueous extract, is often extremely useful in cases in which hyoscine or hyoscyamine have failed, and sometimes in combination with them. The solid opium should be given in doses of 1 grain once or twice a day, and the aqueous extract in a dose of $\frac{1}{8}$ grain, once to thrice daily, but care should always be taken to employ a perfectly reliable preparation of opium, as there are many worthless and cheap specimens upon the market. Morphine I have almost discontinued using for the last few years, because of its unreliableness in this class of the insane. In many cases of this kind the patients will refuse to take medicine, and then it may become important to give it without their knowledge, which can usually be readily done, for the drugs can be easily dissolved in a cup of soup, cocoa, coffee, or any other liquid, or otherwise mingled with the food. In some violent cases it may be necessary to administer the hyoscine, hyoscyamine. or opium hypodermically. In all the acute cases, however, this treatment should, if possible, be supplemented by several weeks of rest in bed, and I know of nothing that will quiet the patient better than this. The chronic cases should be committed to an asylum unless it is possible to guard the community thoroughly against them by proper attendance at home. They are too dangerous to be at large, and the most harmless of them may at any moment do some violent deed, as in the case of the bomb-thrower Norcross, who exploded ten pounds of dynamite in a business office in this city.

CHAPTER XVIII.

PARALYTIC DEMENTIA.

Synonyms: General paralysis of the insane. Progressive general paralysis. General paralysis. General paresis. Paralysis of the insane. Paralytic dementia, or Dementia paralytica. Paretic dementia. Allgemeine progressive Paralyse der Irren. Paralytische Blödsinn. Allgemeine progressive Gehirnlähmung. Paralysie générale progressive. Périencéphalite chronique diffuse. Démence paralytique.

Definition. Paralytic dementia is a cerebral disease of chronic, remittent type, characterized by dementia of very gradual onset, usually merging into mania or melancholia, generally with stupid and expansive delusions, and accompanied by tremor, ataxia, pupillary alterations, and eventual paresis.

History. The first description was given by Bayle in 1822, who considered it to be an arachnitis. From this period until the early '50's the main treatises were by Frenchmen; but in 1854 Erlenmeyer, a German, wrote upon it, and from that time to the present it has been considered by the writers of every civilized nation, a catalogue of whose names would fill pages.

Clinical History. Although many diseases of the brain have been falsely diagnosed as paralytic dementia, and although the future will undoubtedly make it evident that some cases of what we now regard as this malady are really essentially different affections, it is entirely possible at the present day to make a positive diagnosis of the one clinical entity. When we come to consider the pathological anatomy of the disease, we shall find that paralytic dementia consists of molecular alterations that affect the cerebrum as a mass, and this fact should at the outset make us realize clearly that the symptoms, both mental and physical, are co-extensive with the cerebral functions, so that there is scarcely any one symptom to be found in other diseases of the brain which cannot be found in this, differing only in the manner of grouping. Authors are at variance as to the number of stages, but in my experience these are in the main as follows:

The prodromal;
Maniacal or melancholiac;
The demented.

But there are many exceptions to this rule in which only the first and second stages are observed.

The prodromal stage. This may extend over months or years, and the mental and physical symptoms are remittent and insidious. The mental symptoms consist of alterations in character that are often vague and transitory, or of intensifications of pre-existing mental

traits. The patient becomes eccentric, and performs some act that is so causeless and so entirely at variance with his usual behavior as to puzzle those acquainted with him. One patient of mine, for example, who had been newly married, brought home a rose to his wife, gave it to her affectionately, seated himself before his mirror to brush his hair, then, suddenly rising, slapped her face, and nonchalantly went about his dressing as if nothing had occurred, without any word of explanation, seemingly unaware that he had done anything out of the way. Another, who was a cautious business man, went to a firm of brokers and engaged recklessly in speculation. Another became very suspicious of his family without cause, although he had hitherto been of a jovial and kindly disposition. On the other hand, a naturally frugal man becomes penurious; a generous man grows extravagant; one who is reticent becomes still more silent, and a talkative individual is converted into a loquacious one; and thus we witness a deepening of all the innumerable shades of character. At the same time—often, indeed, at the very first—there is a change in the finer ethical sentiments, which lie on most civilized human characters like the bloom upon the peach, indicative of the highest culture, and varying in each individual with the social station, sex, age, race, and nationality. So that a kind father or husband becomes brutal or seeks the society of loose women, or a chaste and modest woman gives rise to remark, or an affectionate son or daughter becomes disobedient, disrespectful, or thoughtless. Contemporaneously with these mental alterations, physical ones are observed, often so slight as to escape other eyes than those of the trained observer. Among the earliest of these are tremor, and speech and pupillary alterations. The tremor is generally in the tongue and in the facial muscles, occasionally also in the extremities. It is best observed in the former by causing the patient to protrude the organ slowly, when the lingual muscles are seen to be agitated by a series of slight fibrillary movements. In the latter the tremor is best seen by causing the patient to speak, when the facial muscles of the lower part of the face, especially at the angles of the mouth, will twitch with slight muscular twitches that are intermingled with tremor. The speech-defects are best made evident either by a slurring over and indistinctness in the pronunciation of certain words or of consonants and labials, or by a combination of these two peculiarities. Frequently, however, a patient who can pronounce individual words distinctly will slur over these same words when he pronounces them in the context of a sentence or in ordinary conversation. The pronunciation of the letter "r" is apt to be especially difficult, particularly to a Frenchman or a German, with whom the "r" is either guttural or prolonged, although this defect can almost invariably be observed even in the English-speaking races. A good test of this is to get the patient to pronounce such a sentence as "riding cavalry brigade," or, better still, the same words in German, "reitende Cavallerie-Brigade," with the full German guttural intonation, or the sentence, "round about the rough and rugged rocks the ragged rascal ran." When the tremor is slight in the extremities it can best be detected

in the hand, by causing the patient to extend the arm at full length at right-angles from the body and then lightly supporting the fingers upon the palm of the examiner's hand, when a delicate muscular tremor will be made evident, like the throbbing of a small engine. Tremor in the lower extremity may be detected by having the patient sit down and extend the lower extremity at right-angles from the body, and then lightly supporting the heel with the examiner's palm, when the foot and toes will be seen trembling with fibrillary tremor that is usually more distinct and coarser than is observed in the fingers. The pupillary alterations are to be noted in the size of the pupils, their marginal contour, and the reaction of the iris to light, consensual or cutaneous stimulation, and accommodation. The pupils in about one-half of my cases have been dilated, my observations thus agreeing with those of Bevan Lewis, and whilst the moderate-sized pupil is next in frequency, the contracted pupil is infrequent. In about one-half of my cases one pupil was larger than the other. These were also about the same figures in Bevan Lewis's patients, but whilst he states that the right pupil was generally the largest, this has not been constant in my cases, as in the same case sometimes the one pupil and sometimes the other would be the larger. This pupillary inequality is a symptom that is very apt to escape observation, inasmuch as it may be temporary, lasting only for a few days or weeks at a time; indeed, in one patient of mine, about whom there was a question of diagnosis, this inequality was not observed by me for upward of some three weeks, and was finally detected by my assistant during my absence from town, but I did not see it myself for several weeks afterward. The pupil may be perfectly immobile or excessively sluggish in reaction to light, tested by a strong light and focal illumination of the eye by a convex lens, and in some cases this sluggish reaction may be followed by wide dilatation; or the pupil may be sluggish, or it may not react with accommodative movements of the eye; or it may fail to respond or respond sluggishly upon stimulating the skin by the electric brush, by pinching, or by a pin. It is usually the smaller pupil which fails to react consensually, or with the light-reflex, and the consensual movements generally occur when there is impairment of the light-reflex, although the light-reflex may be impaired whilst the consensual reflex may be active. The knee-jerk in this prodromal stage is abnormal in a majority of cases, being sometimes exaggerated, sometimes slight, sluggish, or absent. Transient urinary incontinence is also an occasional symptom of the prodromal stage. Whilst all these symptoms that have been enumerated are more or less constant, varying slightly in intensisy, perhaps, there are yet apt to be paroxysmal exacerbations, when the mental defects, the speech-alterations, the tremor, and the pupillary abnormalities are increased, and the face becomes congested or pale, sometimes also the extremities. These exacerbations last from one to several days. As time goes on, these symptoms increase in frequency and intensity, and the ataxia which has constituted the speech defects becomes manifest in the upper extremities. It will best be made evident by getting the patient to

write his name, or by having him close his eyes, swing his arm from his side, and quickly bring the tip of his index-finger to touch the tip of his nose (page 171). The muscular strength at this time is usually unimpaired, as can be demonstrated by testing the strength of the facial muscles or the grasp of the hand; but in some few cases, even in this prodromal stage, the paresis may be observed in some of the muscles of the face, more especially in a flattening or sluggish movement of the muscles making the naso-labial fold, in slight deviation of the tongue, in a loss of power in one eyebrow or one side of the brow, or even in imperfect action of one upper eyelid.

These prodromal symptoms are of vast importance, especially in a medico-legal sense, for it is by means of them that an early diagnosis can be made and much suffering frequently saved to the patient, his relatives, and his business interests. Suddenly or gradually they will merge into the second stage.

In the maniacal or melancholiac stage the patient has superadded to the other symptoms a mania or a melancholia, each with certain peculiarities distinguishing it from other manias and melancholias. In the mania of paralytic dementia there is almost invariably a certain stupidity tingeing the delusions and the hallucinations, so that the former are illogical, and with this stupidity is mingled an expansiveness or exaggeration of idea giving rise to the so-called *delirium of grandeur*. The patient, for example, may say that he owns all the theatres in the United States or all the steamboats, that he will make his physician a present of a million, that he is the most powerful man in the world, and so ramble on in this extravagant strain; but when you ask him how it is possible that he can be worth all this when his circumstances are known to be far inferior to what he claims them to be, he reiterates his assertion without attempting to reason about it. In this respect he affords a marked contrast with the paranoiac who may have the same grand conceptions, but will have any number of settled and fine-spun reasons with which to lend them plausibility. It is this air of stupidity or dementia tinting the delusions and hallucinations that is so especially characteristic of the paralytic dement and gives one of the names to the disease. The mania usually becomes violent, and in some exceptional instances becomes so violent as to lead to a fatal termination by exhaustion. The melancholia of the paralytic dement is not accompanied by obstinate insomnia, or post cervical ache, but there are often suicidal tendencies, and the delusions and hallucinations are the depressed ones characteristic of ordinary melancholia. In some few instances I have observed illusions in paretic dements, but it is very difficult to determine this in such cases. In both the melancholia and the mania paroxysms of terror are characteristic, coming on frequently without external cause.

This second stage, of mania or melancholia, may, however, as I have already said, be wanting in certain cases, in which there is a simple dementia throughout; but if present, it usually merges into the third stage, of *dementia*.

Although there is a certain element of stupidity throughout all three stages in the paralytic dement, this stage of dementia proper is marked by downright lack of mental faculty to a greater or less degree, so that the patient becomes stupid or childish, talking and acting foolishly, at the same time that he may be difficult to restrain temporarily.

Throughout all these stages of paralytic dementia there may occur epileptiform or apoplectiform attacks; *i. e.*, slight loss of consciousness, with or without convulsions, localized or generalized, or losses of consciousness varying from mere syncope to downright coma, with paralysis that may be hemiplegic or monoplegic in distribution or affecting speech. Usually toward the end of the first stage, or during the second, paresis affecting at first the lower extremities begins to appear, showing itself at first only in the paroxysmal exacerbations, and gradually increasing in degree until in the later stages it may become complete paralysis of the upper and lower extremities. In some cases, however, the paretic symptoms are among the earliest, and it is by no means infrequent to find an initial tremor, pupillary alterations, and defects in speech accompanied by a paresis of one side of the face and tongue, or to note that the patient, even at this early stage, has a distinct paresis over and above his ataxia.

Certain of the symptoms have peculiarities that are at times of aid in diagnosis, and which should, therefore, be considered in detail, such as affection of smell, sight, hearing, tact, pain, muscular sense, and temperature.

Voisin claimed that the sense of smell was always defective in the initial stage of general paresis, but in this he has not been borne out by the later writers, and my experience leads me to state unqualifiedly that he is mistaken, for I have yet to see the first case of this disease in which the sense of smell was affected in the early stage, although I have occasionally seen it affected in cases of cerebral syphilis simulating general paresis. In the later stages, as most writers are uniform in stating, it is certainly impaired; but this is rather due to the mental impairment than to any particular lesion of the olfactory apparatus itself.

The sense of sight is sometimes affected in a peculiar way, first described by Fuerstner. The defect is usually in one eye, especially the right. Single letters are recognized, and the patient can name them; but when combined in a word they are not recognized, and it is the same with simple objects. This is really nothing more than a variety of the mental blindness which has been elsewhere described in this work. The ophthalmoscope is of but little diagnostic use in general paresis, for it is in only a small proportion of cases that any lesion is found, generally of the nature of a primary optic-nerve atrophy, and many of these are those which are associated with spinal symptoms, although it can be observed in pure cerebral cases. As the symptom, however, is not usually observed until the disease is well established, it generally has but slight value in the question of differential diagnosis.

Hearing is occasionally over-acute at the onset. Word-deafness (*vide* page 140) is observed also at times.

The sense of pain is generally acute in the first stage of general paresis, and there are apt to be hyperæsthesia and neuralgia. Sander calls attention to the fact that an onset of migraine in an individual in the fourth decade of life, without a precedent history either personal or hereditary, is of great diagnostic value, and I think he is right. Anæsthesia is a usual accompaniment of general paresis when the disease is well marked, and occasionally even in the early stage. In six cases of Mendel's in which there was marked anæsthesia there was also a spinal lesion; but the former has occurred in cases of mine in which there were no spinal symptoms whatsoever, and it is not necessary that there should be such. This anæsthesia in the later stages may become very great. Mickle relates an instance of a patient chewing his right forefinger as if it had been a piece of tough food, so that it became gangrenous and was amputated without any general or local anæsthesia, the patient gazing stupidly at the operation without the least concern or evidence of pain. Baillarger relates a similar case of amputation, and Lines tells how a patient seized a live coal and kept it in his hand long enough to produce a severe burn. I have seen anæsthetical phenomena almost similar to this in several instances, and in one case that had just passed out of the initial stage into an attack of mania the anæsthesia was so complete that a large, jagged wound caused by the patient thrusting his hand through a plate-glass window was dressed without the slightest sensibility being manifested. This tendency to anæsthesia should always be borne in mind when general paretics are made to bathe in warm water, and it is often an explanation of the so-called parboiling accidents occurring in asylums and creating so great a furor in the sensational newspapers of the day. Fulgurant and stabbing pains may, of course, be met with in the cases in which the disease is complicated with tabes.

ETIOLOGY. The causes of paralytic dementia are—

Sex;
Age;
Heredity;
Occupation;
Social position;
Other insanities;
Cranial traumata;
Climate, locality, and race;
Syphilis;
Alcoholism;
Food and other poisons;
Spinal disease.

Women are much less frequently attacked than men, and a careful analysis of statistics made by Mickle has shown that about four males are attacked to one female. It is probable that this difference between the sexes is largely due to the greater exposure and tension of men in life and their greater tendency to dissipation and errors of

hygiene and diet, although it must not be forgotten that the female is throughout life the more viable organism.

It occurs chiefly between thirty and thirty-five, although exceptionally it may occur after or before these dates, sometimes as late as sixty and occasionally as early as six.

Heredity of paralytic dementia has been rare in my experience, although neurotic heredity has obtained in most cases.

The military and naval life are said to be conducive to paralytic dementia. Thus, the disease was frequent among the soldiers of Napoleon the Great, and Mickle found many cases in the regiments of the English Guards.

Mickle states that of 64,642 persons admitted to the insane asylums of England and Wales, 5.91 per cent. of the private admissions were general paretics, whilst of the pauper admissions 8.21 per cent. were such. Laehr found only three cases of paralytic dementia among 786 females of the better classes, and Jung asserts that 31.8 per cent. of the males of the better class were paralytic dements, but only one out of 109 females. In the French army, so Colin maintains, three-fourths of the cases of insanity in the officers were due to paralytic dementia.

There can be no doubt that the ordinary psycho-neuroses of long duration can sometimes merge into paralytic dementia, and this has been especially observed in cases of hallucinatory insanity by Marcé and Calmeil, the former's cases having lasted respectively three, four, five, and six years, and the latter's thirteen. At the same time it must be borne in mind that this causation is rare.

There can be no doubt that cranial trauma acts in certain cases as a cause.

There are many curious facts about climate, locality, and race in the causation of paralytic dementia. It is said to be extremely infrequent in Ireland, many of the large public asylums having no case of it, and only 2 to 3 per cent. of the admissions to the Dublin Asylum consist of this disease, whilst it is almost unknown in the Belfast institution, where, it is stated by Mickle, "the population is chiefly of Lowland Scotch origin, and really of Saxon blood, while the Celts of Wales, Cornwall, and the Scotch Highlands have a considerable share of the disease." Yet the Celts of the south of Scotland are rarely affected by it. In the south of France the disease has increased during the last two generations. It is said not to have been recognized in this country until 1843, and then by Dr. Luther Bell. According to Dr. Workman, there was not a single case in 1853, when he entered the Toronto Asylum; but from January, 1865, to July, 1875, there were 72 deaths. The percentage is different in the different provinces of Germany, in different parts of Belgium, in the counties of England, and in the different States of Italy. In Cuba it is infrequent. In Portugal the percentage is about 3 per cent. of all the insanities. In Constantinople, judging by the institution on the Asiatic side in Scutari, which was visited by Mendel, the disease was relatively frequent, and increasing in number according to the statement of the superintendent. It is stated that in Rio Janeiro, Brazil,

there were 11 cases in 297 insane. In Cuba it occurs much oftener among the negroes than with the native whites. In Australia it is said to be frequent.

In a most excellent and painstaking monograph written in 1889 by Morel-Lavallée and Bélières the subject of the relationship of syphilis to paralytic dementia is most exhaustively considered, and the authors show very decidedly that syphilis is admitted by competent observers in all the countries of Europe to be a frequent cause of this disease, although there is no means of differentiating these cases from others of non-specific origin. These writers also demonstrate very conclusively that there is a disease closely simulating paralytic dementia, and designated by Fournier as syphilitic pseudo-general paresis, which may be induced by focal lesions of the cortical or subcortical regions of the cerebrum, or by syphilomata, causing fusion of the cerebral membranes, and leading secondarily to cortico-meningeal infiltration. Some forty-six cases are carefully collated and detailed to illustrate these conclusions. Although the authors do not offer any tangible means of differentiating syphilitic pseudo-paresis, yet a study of their cases will show that in every case there was a precedent history of cerebral syphilis, and in my cases this was also shown. The duration of the disease may be said to be from three to five years, although cases have been narrated that ended in a few months, and others that lasted many years.

Diagnosis. It should be remembered that general paresis is really a gradual dementia, complicated by stupid delusions, often expansive in nature, by pupillary alterations, by motor symptoms that are ataxic in the initial stage and paralytic in the later, by general tremor that causes a peculiar pronunciation, and by the supervention, in many instances, of a mania or melancholia upon the initial symptoms. The malady is, therefore, really a chronic dementia of a peculiar type, with physical symptoms. The pupillary myosis, or dilatation, or inequality, or reflex alterations, the tremor of the tongue and facial muscles, and the peculiar pronunciation, associated with mental alteration that is vague and stupidly eccentric, the tendency to paroxysmal exacerbations, lasting a day or two or more, in which all the symptoms are exaggerated, and flushing or pallor are superadded—all these make a picture in the early stage that is seen only in this disease, although there may be a certain resemblance to it in others. When to these symptoms are joined delusions of grandeur, the diagnosis becomes very easy; but even when the dementia has not been interrupted by any markedly expansive delusions the diagnosis can be made by consideration of the foregoing facts. When to these symptoms of the initial stage have been joined a mania or melancholia, of gradual or sudden onset, the diagnosis is still more easy, and in every case of melancholia or mania the precedent history should be carefully obtained.

The diseases from which general paresis should be differentiated are—

Disseminated sclerosis;
Paralysis agitans;

Cerebra syphilis;
Primary dementia;
Secondary dementia;
Melancholia or mania;
Katatonia;
Paranoia;
Alcoholism;
Bromism;
Lepto-meningitis cerebri.

The diagnosis from disseminated sclerosis is not always easy, and the diagnosis from a meningitis of the pia mater is sometimes equally as difficult. Usually, however, in disseminated sclerosis the mental symptoms are not so pronounced at the beginning. There is not the same degree of ataxia, there is more apt to be nystagmus and the pupillary alterations characterizing general paresis, and the tremor precedes the mental symptoms by a period of months or years. Nor is there in disseminated sclerosis the tendency to paroxysmal exacerbations which is seen in most cases of general paresis. In disseminated sclerosis, too, the tremor in the early stage is usually of the intentiont-ype, and this is rare in general paresis.

Paralysis agitans is easily differentiated from general paresis because of the peculiar bent attitude, the slow, deliberate speech, instead of the stuttering, scanning enunciation of the general paretic, by the lack of mental symptoms, except some dulness in the later stage, by the lack of marked pupillary alterations other than those belonging to old age, and by the absence of any paroxysmal exacerbations, although cases of paralysis agitans may have apoplectiform attacks.

I feel quite positive, although I am aware that I hold my opinion alone against most authors, that cerebral syphilis can in most cases be diagnosed by means of the symptoms to which I have called attention, namely, quasi-periodical headache and insomnia, both headache and insomnia ceasing upon the supervention of any paralytic or convulsive symptoms; and when a cerebral disease characterized by these symptoms, preceded or not by a history of specific infection, passes into dementia resembling general paresis, the diagnosis can be made in the larger number of instances. In some, however, in which the history of these inititial symptoms is wanting, the diagnosis cannot be made.

Primary dementia occurs in individuals who are younger than general paretics, and the dementia is of an entirely different character, the patient acting sillily and talking incoherently and foolishly, whilst the onset of the dementia is sudden as compared with the very gradual and insidious onset of the first stage of general paresis. It must be remembered, too, that the dementia of general paresis is only vaguely manifested in the earlier stage, whilst that of primary dementia comes on full-blown, so to speak. In primary dementia, too, the ataxia, the pupillary alterations, the apoplectiform and epileptiform attacks, all are wanting.

The secondary dementias are always, as the term implies, secondary

and terminal conditions of different forms of insanity; but the previous history should make it plain in every instance that the previous insanity has not been of the nature of general paresis.

Every case of melancholia or mania should have the previous history brought carefully to the light, so that the melancholiac or maniacal stage of a general paresis may not be mistaken for an initial melancholia or mania.

Katatonia may in some cases be mistaken for general paresis, but a careful examination of the patient and the history will make the differential diagnosis easy. Katatonia is a cyclical disease, passing through a variety of mental phases, interrupted by choreiform, epileptiform, and cataleptoid attacks, and characterized by verbigeration. General paresis, on the other hand, is a chronic dementia of a remittent and insidious type, varied occasionally by demented agitation or demented depression without verbigeration, occasionally with apoplectiform and epileptiform attacks, but seldom or never with cataleptoid symptoms. In katatonia, too, the pupillary inequality and the pupillary reflex-alterations are wanting, as are also the ataxia, the tremor, the peculiar speech, and the eventual paresis.

Cases of paranoia that have expansive delusions may be mistaken for general paresis; but the resemblance between the two diseases is too superficial to lead to more than temporary mistake. The delusions of the paranoiac are always reasoning and logical within certain limits, whilst those of the general paretic are stupid and non-logical. The paranoiac may have occasional tremor of tongue or facial muscles; but so far is he from evincing the dementia of the general paretic that he is abnormally keen and acute, and in him are lacking the ataxia, the characteristic pupillary alterations, the tremor, the tremulous speech, the epileptiform or apoplectiform attacks, and the terminal paresis.

Some cases of alcoholism bear a strking resemblance at first sight to general paresis, especially those which I have found on the post-mortem table to consist of opacities of the pia mater and congestion of the cerebral substance. The diagnosis can be made by means of the alcoholic history, the relatively acute onset of the symptoms, the tendency of the delusions to be keener and more logical than those of general paresis, and to partake of the character of suspicion, especially of marital infidelity. It must be borne in mind, however, that prolonged alcoholism of itself produces a genuine general paresis, and that these cases of which we are now speaking are simply cases of acute alcoholism.

Bromism may, to a careless observer, simulate general paresis; but the distinction can be readily made by obtaining the history, by the fetid breath, by the acne, by the excessive stupidity without delusions, by the lack of any other pupillary alterations than dilatation, by the lack of ataxia and tremor, by thick, muffled speech rather than a tremulously scanning one, and by the early onset of general weakness that may simulate paresis.

Prognosis. The prognosis of general paresis is a very serious one, although undoubtedly competent observers, such as Minot, have

put on record cases of cure. I have never seen one, but I have witnessed remissions of considerable duration, and it should always be borne in mind that these may occur in any case.

PATHOLOGY. We are just beginning nowadays to obtain a clear insight into the pathological alterations of the cerebral tissues in this disease. Until recently striking results of morbid alterations have been described and have passed as explanatory of the textural changes, although they have in reality been the terminal results of a long series of microscopic alterations that have themselves eluded our ken. This has been due, in the main, to two causes: first, our lack of complete knowledge of the histology of the cerebrum; and, second, to our defective hardening-methods. The marvellous bichromate of silver stain, first discovered by Golgi and subsequently applied by the genius of Cajal (*vide* pp. 17 et seq.), has now, however, revolutionized our knowledge of the marvellous mechanism of the brain; whilst the method of so-called "control" experiments—*i. e.*, investigations of normal tissues by the same method—has brushed away many descriptions of deceived investigators. There is a difference of opinion among cerebral pathologists, for instance, as to whether paralytic dementia primarily begins in the bloodvessels and their lymphatic appendages, or whether its onset is to be found in the nerve-fibres and the nerve-cells. As a matter of fact, this is an idle strife of words, because there have been put on record no pathological observations capable of proving that all the fine medullated nerve-fibres of the cortex, such as are described by Cajal and his followers, have been thoroughly examined, for the simple reason that the staining-fluids used in the pathological cases will not stain all this network of fibres, and it is therefore impossible to say that authors who have described only vascular lesions in the early cases may not, however conscientious and truthful they may have been, have overlooked certain degenerations in these nerve-strands. Then, again, Binswanger has demonstrated that all the hardening-methods alter the original structure of the cell, whilst Frommann positively maintains that the so-called conservative solutions, such as absolute alcohol and chromic acid, often entirely destroy these bodies. Nor is this all, for certain alterations described as pathological have been shown to be due to immediate post-mortem change. Thus, Zacher describes with great particularity certain microscopical details in nerve-fibres, which he would have us regard as the earliest signs of the disease, such as irregular nodules, varicosities, brittleness, normal axis-cylinders shown through transparent medulla, atrophy, irregular curved shape, and final disappearance, observed especially in the tangential fibres and the sublying strands; and yet Binswanger has ruthlessly demonstrated the same appearances in the brain of a man who had been beheaded! In spite of the difficulties of the histological technique, however, it can now be said that the changes affect:

1. The nerve-fibres;
2. The nerve-cells;
3. The neuroglia and the vessels;
4. The membranes.

In 1880 Tuczek thought there was a progressive disappearance of nerve-fibres from the anterior, basal, medial, and lateral portions of the cerebrum, as far back as the anterior central or ascending frontal convolutions, the gyrus rectus, at the base of the brain (Figs. 14 and 18), being also implicated in some acute cases. The fibres involved were those which are known as the tangential fibres of the cortex, as well as the superradiating and interradiating fibres. (See Fig. 29, page 53.) In these cases, in addition to a decrease or disappearance of the nerve-fibres, there was an increase or fibrous transformation of the interstitial tissues, with an abundant development of abnormally large spindle or Deiters' cells. Tuczek claimed that these pathological appearances were pathognomonic of Paralytic Dementia, and he holds this view to the present time; but whilst the facts to which he first called attention have been abundantly confirmed, his interpretation of them has not met with the same happy fate, as the same fibres are frequently wanting in other forms of mental disease. Thus, Zacher, examining the brains of twelve paralytic dements, endorsed Tuczek's statements; but in three brains of ordinary paralysis, two of paranoia, and three of senile dementia, he found the same peculiar disappearance of nerve-fibres, as did Keraval and Targoula in secondary dementia, Emminghaus in post-febrile dementia, Jendrassik in tabes dorsalis without mental impairment, and Greppin in several cases of what he calls post-melancholiac dementia; whilst Kostiurin, examining the brains of twelve perfectly sane people, varying in age from sixty-five to eighty-eight years at death, observed a considerable loss of tangential fibres in the parietal lobe. Greppin, moreover, could not find any lack of the intracortical nerve-fibres in a case of paralytic dementia of nine months' duration. Indeed, Fischl points out that there are great physiological differences in the number and calibre of the cortical nerve-fibres, and that a sufficient regard has not been paid to these physiological conditions. Binswanger, who has lately made the most thorough study of the pathological data about this disease, believes that it is only possible to reach conclusions as to the disappearance of fibres in the uppermost, or tangential layer (Fig. 29, page 53), and the interradiating fibres, whilst the findings in the superradiating fibres are uncertain; but there can be no question that the bloodvessels are implicated at an early stage, although, as has already been said, there is a doubt as to whether they are at first affected. These changes in the vessels are in the nature of a hyaline degeneration. By them is induced a fluctionary and passive hyperæmia, and the outflow, through the wall of the bloodvessels, of numerous red and white blood-cells. Probably because of a certain amount of destruction of tissue, as well as of the transudation, there soon follows a dilatation of the extra-vascular lymphatic system, and the veins become distended with red blood-cells, although, curiously enough, white blood-cells have not been observed within them.

The alterations in the basic and connective-tissue substance of the brain, as well as in the vessels, have been of great interest, inasmuch as upon the termination of them rests the question as to whether the

disease is really primarily an interstitial encephalitis. At the outset there is great question as to how much of what was formerly regarded as basic and connective-tissue substance was really nerve-processes which have been shown to exist by the newer methods. Different authors have attached different meanings to the increase in the number of free nuclei, the appearance of spindle-cells, the alteration of the basic substance into thickened and fibrous tissue, and the proliferation and thickening of the neuroglia. The older writers regarded these appearances as due to a true inflammatory process, beginning in the bloodvessels, the nerve-elements being terminally implicated. In an elaborate paper upon this subject, as far back as 1880, Binswanger demonstrated that there were varieties of nuclei coming from the different structures of the cerebrum, viz., from the smooth muscles of the vessels, the neuroglia, the ganglion-cells, the adventitial sheaths, the pericellular spaces, and also certain free nuclei which are found strewn throughout the cerebrum. Some of these color readily, whilst others do not, the former being generally the smallest. These nuclei are irregularly distributed in the white and gray substances, vary greatly in number in different individuals and in different portions of the cerebrum, and are more numerous in advanced age. In addition to these facts, investigations by Lubimow, Witkowski, His, Gierke, Golgi, Cajal, Kölliker, and Retzius, have only served, as has already been said, to create great doubt as to what the so-called basic and connective-tissue substance is, and this uncertainty has naturally reflected itself in the observations as to the nature of paralytic dementia; as a consequence of which nothing more can be definitely said than that the neuroglia is sometimes atrophied and sometimes proliferated, whilst the spindle-cells in the middle and deeper layers and the cortex are not increased in number except in the later stages.

Cionini has found the gray matter of the cortex diminished in thickness, especially in the central convolutions and the frontal brain, partially also in the occipital lobe. It would therefore appear that a certain amount of cortical substance must have been destroyed, and what has remained has become more compressed, whilst the physiological spaces around cells and bloodvessels have widened.

At the same time there begins an active proliferation (generally regarded as reparative) in the endothelial adventitia of the arterioles, capillaries, and veins, leading to a thickening of these structures and proliferation of the endothelial nuclei. A portion of these new formations also undergo a hyaline degeneration at a later stage. In some vessels the process of proliferation even implicates the true wall of the vessel. By these means the lumen becomes narrowed, and in later stages even obliterated. A new formation of vessels is doubtful, although Bevan Lewis thinks he has seen it. The intra-adventitial lymph-spaces are only affected in places, separated from the media, and containing red and white blood-cells, coagulated lymph, and amorphous and finely granular pigment; but white blood-cells are only found occasionally; indeed, the presence in large masses of the latter is regarded by some authors, notably by Binswanger, as an

indication of the course of the disease, for he believes that they are seldom found in the intra-adventitial lymph-spaces in large number in slowly progressive cases with paralytic intercurrences, whilst they are present in those with chronic and frequently repeated venous congestions. The extra-adventitial lymph-space, together with the pericellular space, is very generally dilated. In the former is also found, though in a less degree, white and red blood-cells, fibrinous coagula, and pigment; but in the lymph-spaces of the cortical substance itself, as well as in the pericellular spaces, no distinct increase of the lymphoid elements has been observed. True inflammatory or exudative processes may be added to these alterations with the further progress of the disease, and they are demonstrated clinically by somnolence and irritative and paralytic phenomena; and if death then occurs, there may be marked infiltration of both the extra- and intra-adventitial lymph-system, although it has been demonstrated, on the other hand, that these inflammatory changes may be wanting, the clinical phenomena then being probably due to the obstructed flow of the lymph-stream produced by the dislocated cerebral pressure. In the later stages of the disease the pia mater plays an important part. In the early stages this membrane is only moderately affected over the convexity of the hemisphere, particularly in isolated places in the frontal lobes, being thickened and proliferated in its endothelial nuclei; but with the course of time the thickening and nuclear proliferation of the endothelial adventitia of the other vessels extend to the similar structures in the pia; and these hypoplastic processes of the pia are regarded as an extension of the proliferation of the vessel-sheath to the endothelial membrane. Partial adhesions of the pia to the connective tissue lead in places to imperfect obliteration of the epicerebral spaces, this being observed in the region of the vessels going to the cortex, as well as in the portions of thickened pia; and this is due to an increased growth of the pial connective fibres and of the endothelial septa coming from the pia and traversing the epicerebral space. There are thus induced an obstruction in the flow of the lymph-stream, and a stasis in the lymph-spaces of the connective tissue, as well as those in the deeper portions of the cortex. It is not difficult to understand that this altered lymph thus obstructed in its flow should coagulate cerebral tissue which has been already destroyed. These changes do not seem to differ in kind, whether the case be chronic or relatively acute, except in the rapidity of their progress. A certain number of the violent cases are fatal because of the exhaustion produced by the great motor excitement, the deficient nourishment, the pulmonary or intestinal lesions, etc., rather than because of any intensity in the pathological phenomena of the cerebrum, and, indeed, in many of them true inflammatory phenomena are entirely wanting. These alterations in the tissues especially affect the frontal brain, the convolutions of the island of Reil, and the parietal lobe, and finally extend to the temporal and occipital lobes.

Discussion of the alterations in the cells has not been had in this general description because of the uncertainty regarding them.

The ganglion-cells of the cortex, especially in the frontal lobe,

often possess two or more nuclei, one of them, however, being probably the nucleus of a wandering leucocyte. The body of the cell is jagged in places, the space thus formed being filled with the nuclei of leucocytes, whilst the pericellular spaces are widened, and contain one or more leucocytes, as well as pale, ovoid nuclei which are coarsely granular. What these large ovoid cells are is uncertain, although Binswanger believes that they come from the endothelium. They have often been mistaken for the true nuclei of the ganglion-cell. Many other alterations in the cells have been described by different authors. Thus, Binswanger states that the nucleolus is often swollen, and contains peculiar white, glittering, irregular lines, producing a cracked appearance; or the place of the nucleoli is taken by two to four irregular lump-like large granules, or the nucleus holds a large number of small, dark, distinct granules, whilst the body of the cell is unaltered. In other cases the same author has seen the nucleus enlarged, puffed, cloudy with granules, or holding glittering plates; or its contour is irregular, or merges with the surrounding cell-protoplasm, in both of which instances the nucleolus is unaltered. In other cases the body of the cell appears normal, but the place of the nucleus has been taken by a loose, finely granular mass, and in still other cells the nucleus has disappeared, being replaced by brown pigment, and the nucleolus is also gone. With these alterations of the nucleus and the nucleolus, the body of the cell is irregular, bellied, diminished, darkly pigmented, and contains small nuclei without nucleoli. Alongside of many cells thus altered are found others perfectly intact. These cellular alterations were observed in the central, the third frontal and neighboring convolutions, and those of the island of Reil. These observations of Binswanger have been confirmed by Liebmann, Zacher, and Bucelski; but in certain investigations undertaken by Fischl he found that in some hardening-methods, notably in Flemming's, Ehrlich's, or Müller's solutions, the nucleoli of the cells would disappear or diminish, the place of the nucleolus would often be taken by pigment, and its shape would be altered and the nuclei would lose their sharp outlines, whilst pigmentary collections would almost constantly be found in the ganglion-cells. Nevertheless, as pathological he regarded pigmentary degeneration with fattiness and entire destruction of the ganglion-cells, and the nuclear and nucleolar alterations found in normal brains are seen in a larger number of cells and in many more places in pathological instances. Fischl was never able to obtain the hyaline degeneration of the cell-body (spoken of by Liebmann and Lubimow) in alcohol preparations. Binswanger has found the cortical cells diminished in number in the early stages and in the galloping cases, although this was not very marked, and the cells were often only affected singly, and in individual groups in the frontal lobe and the central convolutions. Friedmann has made some very interesting observations upon the early alterations in the ganglion-cells of the anterior horn, and he states that the death of the cell is effected in several ways: (1) By a homogeneous swelling of the cell, which is really a hyaline

transformation of it, commencing in the centre, whilst a small peripheral portion of the cell may remain intact for some time, containing the chromatic substance, and even the nucleus, which latter disappears late, but yet before the nucleolus ; (2) by a granular destruction or fatty degeneration. In this the diseased portions are transformed into a pale, granular mass, as the chromophile substance disappears. The first step in this type is the granulation, and not the fattiness. Types 1 and 2 are often combined ; (3) by sclerotic degeneration, which is less frequent than the other forms in the acute inflammatory period, whilst it is more often found in the chronic cases ; (4) by the transparent light-cell. This is a very infrequent form, characterized by the destruction of all but a small portion of the chromatic substance, the nucleus often remaining intact. This author does not believe in the existence of simple atrophy or shrinking of the cell, or in the so-called cloudy swelling ; the first being, according to him, only the result of the homogeneous degeneration, and the latter a combination of swelling with the second stage of the molecular destruction. These observations, however, have not yet been confirmed. They have led, however, to a great doubt as to whether the active inflammatory changes in the ganglion-cells described by others really occurred, inasmuch as they have probably been due to defective hardening-methods or faulty observation.

From these descriptions it can be seen that the theory of paralytic dementia being due to an interstitial encephalitis, as was maintained by the older authors, is not warrantable. Nor is there any confirmation of the views of Bevan Lewis, who lays great stress upon the *rôle* played by the connective tissue, for he would have us regard the connective-tissue (or spider or Deiters') cells as playing the part of depurative agents, being, as he expresses it, true *scavenger*-cells. He maintains that these bodies are auxiliary to the capillaries, that they feed upon the degenerated nerve-cells and fibres, both of which are converted into granules or oily droplets, and he even presents many alterations of the condition of these scavenger-cells at different stages of the degeneration of the cortex, figuring them laden with the products of the disintegration of nerve-cells and nerve-fibres, and actually at times drawing upon the perivascular sheath with such force as to distort it. Nor, in view of these newer investigations, need we attach more than a relative importance to certain gross phenomena which have been observed in the brains of paralytic dements. Thus, meningeal hemorrhage may occur on the outer surface of the arachnoid into the subdural spaces, and it may be a mere streak of fluid staining, a coagulation, or a rust-stained pellicle that may be peeled off, forming one of the varieties of the so-called arachnoid cysts. Fuscous degeneration of the cortical nerve-cells may sometimes occur. Spitzka has described a cystic degeneration of the cortex found either in the gray matter or in both the gray and white, consisting of minute cavities, varying in size from a pin-point to a millet-seed.

TREATMENT. The first point to determine in any case of general paresis is whether there has been a history of cerebral syphilis, and then whether it is possible to cause improvement of the symptoms by large and increasing doses of the iodide, or, if the iodide is not well borne, by the conjunction with it of mercury. (See "Syphilis of the Nervous System.") If there should be no cerebral syphilis, the use of either iodide or mercury will be of slight value, simply diminishing the tendency to convulsive and apoplectiform attacks and to agitation; but in this event the dose need not exceed 15 or 20 grains three times a day. The use of large doses of ergot has sometimes seemed to me to affect the disease favorably. The fluid extract should always be employed, commencing with $\frac{1}{2}$ drachm doses three times a day, and increasing to $\frac{1}{2}$ ounce three times a day if the patient's stomach will carry such a quantity. Sulphate of quinine, 5 to 10 grains at bedtime occasionally, with 20 grains of bromide of potash, has also seemed to me to be of considerable use. In the attacks of mania the patient can be quieted by means of warm baths, by hyoscyamine, by the bromides, and by tincture of veratrum viride. In using warm baths care should be taken to test the temperature thoroughly and not allow it to exceed 100° or 105°, and the patient's sensations should never be relied upon, as there may possibly be the anæsthesia which has been spoken of, whilst too warm a bath may easily lead to vesication. The crystallized form of hyoscyamine should be used — Merck's preparation being the best in my opinion—and the doses should vary from $\frac{1}{300}$ to $\frac{1}{100}$ grain once or twice a day. I do not believe, however, in the large doses, $\frac{1}{50}$ to $\frac{1}{25}$ grain, that are sometimes recommended. The combination with a bromide, the bromide of potassium best, will increase the sedative effect of hyoscyamine without increasing its depressing influence, and 10 or 15 grains of the bromide will answer for this purpose. The tincture of veratrum viride is sometimes of excellent use in quieting maniacal excitation, in doses of 5 to 10 drops; but it should always be carefully watched, and should not be given in cases of cardiac lesion. Electricity I have never found of the least use in cases of general paresis, either the galvanic, faradic, or static current.

CHAPTER XIX.

DEMENTIA PARANOIDES.

Definition. A form of insanity characterized by confused ideas of persecution and self-exaltation without marked emotional disturbance, rapidly passing into dementia.

Clinical History. This is a disease to which attention has been called by Kraepelin, and is well worthy of being known because of its resemblance to paralytic dementia.

The majority of cases begin with confused ideas of persecution, and these are ushered in with prodromata of malaise, insomnia, restlessness, and indisposition to work, but these are often exceedingly vague. They last for several months. During this period of time there is a gradual mental alteration denoted by eccentric and causeless acts, which are often startling in their disregard of conventionality or even morality. Delusions of exaltation or grandeur next make their appearance, and, like those of paralytic dementia, they are stupid and illogical or unsystematized in character. Delusions of hearing are also frequent. The patient, however, has no emotional disturbance, but speaks of his delusions and his auditory hallucinations with perfect calmness. The patients are usually silent, and speak briefly. They will often seem to have a dull consciousness of being sick, especially in the beginning of the disease, and this at times seems to be of a hypochondriacal nature, as they complain of weakness, trembling in the limbs, and difficulty in thinking, but the real appreciation of their condition is never present. In some cases the delusion of grandeur is most marked, in others that of persecution. The delusion of persecution is usually of a startling nature, such as that the patient has had his head cut off by the physician, or that somebody has opened his abdomen, or that mephitic odors have penetrated his room at night, etc., and these lead often to violent assaults upon the persecutors. When the disease is well established, hallucinations are frequent, and involve the various senses of sight, hearing, etc. Although the intelligence may at first be only slightly impaired, the dementia soon supervenes and progresses rapidly. The mood at this time is always an exalted one, and at night the patients are frequently excitable. There are, however, no corporeal or so-called somatic symptoms whatever. The progress of the disease is toward incurable dementia. The age of the patients is generally between thirty and fifty, two-thirds being in the former period of life. The dementia, according to Kraepelin, is worse in the younger cases.

Prognosis. The prognosis of these cases is always unfavorable, according to Kraepelin.

Diagnosis. Dementia paranoides is characterized by the gradual supervention of illogical and stupid delusions of persecution and exaltation, without emotional disturbance or somatic symptoms, and the rapid passage into dementia with marked hallucinations of sight, hearing, and the general senses.

Primary dementia is to be distinguished from it by the fact that the dementia is a silliness not characterized by delusions of persecution or self-exaltation, and the delusions are feeble, often difficult to make out, and the dementia is entirely curable.

Paranoia is easily differentiated by the exquisite logicality of the delusions of persecution and exaltation, by the absence of dementia, and often by the changeability of the delusions. Dementia paranoides, however, may be mistaken for paranoia in the initial stage of depression or exaltation.

From paralytic dementia, dementia paranoides is to be distinguished by the earlier age of the patients affected by it, by the absence of somatic symptoms, the greater fixity of the delusions, and the longer course.

Hallucinatory insanity is demarcated by the fact that there is in it no such dementia as in dementia paranoides, although certain progressive forms of the former disease may be practically indistinguishable from the latter; indeed, Kraepelin does not feel sure that there is not some close relationship between the two types of insanity.

Pathology. Nothing whatever is known of the pathology of dementia paranoides.

Treatment. The treatment is unknown, according to the describer of the disease, Kraepelin.

CHAPTER XX.

DELIRIUM.

Definition. Delirium is a dream-like mental condition with excitement, with or without delusions, hallucinations, and illusions, and occurring during or after some febrile condition or from the action of a poison.

Clinical History. In many cases of delirium there are prodromata consisting of restlessness, mental confusion, reflex excitability to sensory perceptions, irritability, and a mild grade of insomnia, with disturbing dreams. Hallucinations and delusions may then appear, at first fragmentary and vague. These hallucinations are especially prone to affect the senses of sight and hearing, and their variety is very great. Under the influence of these false perceptions, possibly sometimes from other causes, the patient becomes more and more restless and excited. If the grade of the delirium becomes greater, these phenomena become intensified, and in the severer types the mental disturbance may become very great. In other cases the onset is very sudden, and it may even be without prodromata. In still others the delirium throughout is of a low, muttering, so-called typhoid type.

Etiology. The causes of delirium are to be found in all the febrile affections, such as pneumonia, bronchitis, pleurisy, peritonitis, the exanthemata, typhoid fever, typhus, etc.

Prognosis. The prognosis of delirium will depend largely upon its cause, the duration of its causative affection, and the condition of the patient. Taken by itself, however, in any case the delirium adds to the gravity of the patient's condition. In a very general way it may fairly be said that the prognosis is better the more in proportion the delirium is to the degree of fever and the better the general condition of the patient. Moreover, the mild delirious conditions which occur occasionally after typhoid, peritonitis, and some other febrile affections are usually of good prognosis, provided the patient's general condition is good, even when they continue for weeks, and although they may occasionally pass into such other types of insanity as acute dementia and hallucinatory insanity. The prognosis of similar conditions after typhus and variola, however, is said to be unfavorable. With these exceptions, the continuance of the delirium beyond a week, however, is unfavorable, as is also its persistence after the defervescence.

Diagnosis. The diagnosis of delirium is usually made with ease by the history of the case and by the presence of the characteristic mental condition. It needs only to be differentiated from :

Transitory fury, or furor transitorius ;
Epileptic insanity ;
Delirium grave.

Transitory fury has a very acute onset, usually without prodromata or very slight ones, and the condition is, as the name denotes, one of absolute blind, reckless fury, lasting from a few minutes to a few hours, and being succeeded by a deep sleep, without any memory of what has occurred. It is therefore an entirely different picture from that of delirium.

In epileptic insanity there may at times be a delirious condition simulating a delirium very closely in its dreaminess. However, the history of the epilepsy will make the diagnosis easy when it can be obtained. In certain ambulance cases, however, it may be impossible to obtain this history for the time being, whilst the mental condition may continue for several days, rarely, however, as long as a week; and under these circumstances time alone can determine the matter.

In delirium grave there is an alternating condition of furious maniacal violence with absolute intermissions, and this is entirely different from the continuous dreamy condition of delirium.

In cases of muttering delirium, sometimes indeed in those of a more excitable type, it is well to search carefully for any lurking lesion in the pelvic, the aural, and the cranial cavities. I have known of a number of cases of mistaken diagnosis which close examination showed to be due to a pelvic abscess, an aural trouble, a thrombosis of the cerebral veins, or an intracranial abscess.

Treatment. The treatment of delirium should depend, of course, largely upon its cause. If it is possible to remove this, it should always be done, as in the case of a pelvic, aural, or intracranial abscess, or by the opening of a thrombosis of a cerebral sinus. In the febrile affections the treatment of the disease causing the fever is all that can be undertaken. When the delirium is proportionate to the rise of temperature special measures should be adopted if possible to bring the temperature down, and this should be done over and above the general treatment of the case. The measures employed for this purpose should be the cold pack, the water-coil, and the antipyretics. The cold pack and the water-coil are dangerous measures except in robust individuals, and should be used with caution or not at all. For my own part, I usually prefer them only as a last resort in cases in which the antipyretics have failed and in which also it is evident that the delirium rises and falls with the temperature-curve. The best antipyretics are the coal-tar preparations, and of these the least dangerous is acetanilide in doses of 4 grains twice or three times daily. Even in using these the pulse should be very carefully watched, and if it is seen to flag, alcohol and cardiac stimulants should be immediately employed. Whiskey is the best of alcoholic stimulants, and it should be given with a free hand in whatever doses may seem necessary, varying from two ounces to as much as 12 ounces in the twenty-four hours. The condition of the pulse should be the guide as to the dose, as should also be the previous habits of the patient, for those who have been accustomed to alcoholic stimulants will of course take more in sickness than the temperate. If the alcoholic stimulant does not properly affect the pulse, the sulphate of strychnine or digitalis should be administered. The

strychnine should be given in doses of gr. $\frac{1}{30}$ *pro re nata;* that is, three times in the twenty-four hours, or every two or three hours. In some cases, however, in which the strychnine induces disagreeable griping of the intestines, digitalis may be used, either in the form of a reliable fresh tincture, ten drops *pro re nata*, or one grain of the solid extract, or three drops of a fluid extract, or digitaline. The nourishment in all these cases should be pushed as much as possible. Of course, it is impossible to give exact directions for every case, because these will vary so much. In all cases, too, great care should be taken to have the bowels move freely at least once a day, and a good plan is to give $\frac{1}{10}$ grain of calomel every hour or two until the bowels do act properly. The condition of the skin should be carefully looked after also, and it should be kept moist and gently perspiring. Cerebral sedatives should never be used in delirium if they can be avoided, for they are always drepressing, and they very often eventally increase the mental trouble. When it is absolutely necessary to give them, as from the extreme restlessness of the patient, they should be administered very carefully. The best are the bromide of potash and the hydrobromate of hyoscine, the former in doses of 20 or 30 grains, the latter $\frac{1}{100}$ grain. These may be given either alone or in combination, always according to the varying condition—indeed, it is a good rule, when possible, for a physician never tq order a dose without having satisfied himself of the effect of the previous one. I have seen more than one case of perfectly curable delirium depressed into a fatal condition by the reckless use of these cerebral sedatives.

When the delirium occurs after defervescence or in the stage of convalescence of the febrile affections the keynote of treatment is combined stimulation, rest, and nourishment. The warning that has already been given against cerebral sedatives should be repeated here with double emphasis, and it may truly be said that it is very seldom necessary to use either bromide of potash or hyoscine except occasionally at bedtime, to secure a restful night, and even then these drugs should be given in combination with some alcoholic or cardiac stimulant, such as an ounce or two of whiskey or $\frac{1}{30}$ grain of strychnine. Much more useful, however, is the administration of quinine, iron, malt-extract, and food. The following prescription is one that I have found so useful in these cases as to make it almost a matter of routine with me to prescribe it:

℞.—Quin. sulph.	gr. ij.
Ferri. sulph. exsic.	gr. j.
Strychniæ sulph.	gr. $\frac{1}{30}$.

M. ft. capsul. no. j. Mitte xv.
S. One three times daily after meals.

If the iron in this prescription shows a tendency to constipate, for it may be substituted four grains of ferratin. Should the constipation still persist, gr. $\frac{1}{10}$ of aloin may be combined with each dose. The malt-extracts should be given regularly after each meal in full doses. If the patient does not dislike the peculiar taste and odor of

a fresh malt-extract, it is always best to give one of these, as they are rather more reliable than the imported alcoholic preparations. The food should be of the most nourishing description, and if there is difficulty in taking full meals it should be given frequently in smaller quantities. The rest should be absolute, most or all the time being spent in bed until the delirium has entirely disappeared. Again and again have I seen relapses brought on in these cases by making patients walk or taking them out to drive before they had fully recovered.

CHAPTER XXI.

IDIOCY.

Idiocy is a condition of congenital mental defect that is technically distinguished from that of later years, which is known as dementia. The symptoms are simply those of lack of development, and this may vary very much in degree, from downright lack of mind to different conditions of imperfect mental activity, so that one idiot may sing well, another play like a master, as did the celebrated Blind Tom, or another may be startlingly weather-wise, or another may even be a clairvoyant, and so on. The causes of idiocy are chiefly those lesions which have been described in the chapter upon the cerebral palsies of childhood, namely: porencephalitis, hemorrhage, traumata, etc. Besides these there may be other causes: imperfect development of the cerebrum and cerebellum as a whole or in portions; lack or imperfect development of the corpus callosum; hydrocephalus, congenital or acquired; local or diffused encephalitis, which may be primary or secondary to acute processes; meningitis; and in some rare cases cerebral hypertrophy. Besides all these causes Lannelongue has advanced the idea that many more cases of idiocy than have been supposed are due to premature ossification of the fontanelles and synostosis. For many years this supposition has been advanced by different pathologists. Virchow and Welker have supposed that many abnormalities in the shape of the skull might be due to lack of development of the bony tissue from an inhibition due to inflammatory changes in the sutures, whilst Gudden has assumed that it might be caused by insufficient nutrition of the cranial bones from early obliteration of their nutrient vessels. Meyer has demonstrated that in some cases rhachitis can cause the same defect. If the lack of development of the skull is bilateral and symmetrical, so-called microcephaly results. If the vertex of the skull is undeveloped, whilst the basal bones attain their proper size, the so-called Aztec type of skull results, a variety of microcephaly. Gratiolet has pointed out certain cases with a very small skull of thick bones and synostosis at the vertex, whilst the cranial bones at the base were mainly cartilaginous, the petrous bone and the ethmoid being larger than normal, and the fossa of the cerebellum being unusually capacious; so that the cerebellum, medulla oblongata, and the spinal cord were largely developed, while the surface of the cerebrum was scarcely more convoluted than in the ourang-outang. Idiots of this class are extremely vivacious, and Griesinger compares them to birds. These cases have a pointed nose that is beaked, and small, low, and short heads. In the cases of cretinism that are seen endemically in the Alps of Switzerland there is frequently found to be early ossifica-

tion of the cranial bones, and in these the nose is sunken and broad, the eyes wide apart, the cavities of the eyes broad but not deep, prominent cheek bones and jaw as in the so-called prognathism. With these different varieties of arrested cranial development often goes lack of development of the bones of the body and limbs. Lannelongue, however, asserts that it is the premature ossification of the fontanelles, especially, that is the cause of many cases of idiocy, and he has therefore proposed that the skull should be liberally removed over each hemisphere so as to allow abundant space in the development of the cerebrum. This operation is known as craniectomy. The objection to Lannelongue's view is that, as has been seen, arrest of cerebral development producing idiocy may result not only from pathological lesions in the intracranial contents themselves, but also from lack of bony development in the bones at the base, and in either of these cases it would be worse than useless to remove the skull at the vertex. In the second place, the operations that have been done upon cases of idiocy at Lannelongue's suggestion have not shown any beneficial results. Dr. R. S. Newton has collected for me thirty-nine cases in which Lannelongue's operation has been done. Temporary improvement is often seen, and the overwrought minds of parents and relatives are very apt to exaggerate the amount of this; but no case of permanent improvement has been put upon record. Nevertheless, I think that an operation is warrantable in suitable cases. If, for instance, a child has developed up to a certain age, and has then gradually ceased to do so, and if there have been no symptoms of an intracranial lesion, or if the shape of the skull is such as to indicate a deformity such as we have described as being due to lack of development of the cranial bones of the vertex, then I believe that it is fair to perform a craniectomy. Even then, however, it should be borne in mind that a porencephalitis may have occurred without marked symptoms. Even if it does no good, the operation should do no harm, and as there is a possibility that the future may enable us to diagnose suitable cases, I can see no objection to the procedure; but a craniectomy, even when done antiseptically, is a very dangerous operation, as the childish brain does not stand the shock of the operation nearly as well as the adult cerebrum, so that the number of deaths has been large from this cause. The operation must therefore be regarded as one of last resort.

GLOSSARY.

Aba'sia. Unsteadiness of gait. From *a*, privative, and *βασίς*, a step. See *Astasia abasia.*

Acromeg'aly. A disease characterized by enlargement of the extremities and of the face. From *ἄκρον*, extremity, and *μέγας*, great.

Æsthesiom'eter. Instrument for measuring the degree of tactile sensibility. From *αισθησις*, sensation, and *μέτρον*, measure.

Agorapho'bia. Fear of open spaces. From *ἀγορὰ*, market or open place, and *φόβος*, fear.

Agraph'ia. Loss of ability to write. From *a*, privative, and *γραφω*, to write.

Allochei'ria. A perversion of sensibility, in which an impression made on any part seems to be felt on the other side. From *ἀλλος*, another, and *χειρ*, hand.

Amyot'rophy. Atrophy of a muscle. A form of lateral sclerosis is called *amyotroph'ic*, on account of the progressive muscular atrophy characteristic of it. From *a*, privative, *μυς*, muscle, and *τροφη*, nourishment.

Anæ'mia. A condition of debility characterized by diminution in the ratio of red corpuscles in the blood. From *αν*, privative, and *ἁιμα*, blood.

Anæsthe'sia. Privation of sensation; paralysis of sensibility. It may be general or local. From *αν*, privative, and *αισθησις*, sensation.

Analge'sia. Absence of pain. From *a*, privative, and *ἀλγος*, pain.

Anapeira'tic. Relating to spasms of inco-ordination in muscles caused by repeated exercise, as in writers' cramp. From *ἀναπειράομαι*, to attempt again.

Ank'le clo'nus. A series of uniform spasmodic contractions of the ankle-joint produced by continuous stretching of its extensor muscles. From *κλονος*, tumultuous movement.

An'ode. The positive electrode or pole of a galvanic battery. From *ανα*, upward, and *ὁδος*, a way.

An'sa lenticula'ris. Tract of nerve-fibres extending from crusta to lenticular nucleus, and passing under the optic thalamus. From *ansa*, a loop; *lens*, a lentil.

Anthropoph'agy. Cannibalism. From *ἄνθρωπος*, man, and *φάγω*, to eat.

Anticy'clone. A period of blue skies, bright sunshine, high barometer, and slight atmospheric motion. Painful forms of neuralgia occurring especially at that time are called *anticyclon'ic.* From *anti*, opposite to, and *cyclone* (*κυκλος*, a circle).

Ape-hand. Complete atrophy of the thumb-muscles, noticed in progressive muscular atrophy.

Apha'sia. Impairment of or loss of speech, resulting from apoplexy,

embolic softening of the brain, neoplasms, or other cause. From *a*, privative, and *φημι*, to speak.

Arboriza'tion Tree-like appearance of nerve-fibres, as in the cerebrum and cerebellum. From *arbor*, tree.

Arc'uate fi'bres. Fibres in the medulla oblongata which pass from the anterior median fissure to the olivary body and cerebellum. From *arcus*, arch.

Argyll'-Rob'ertson pu'pil or *sign.* Small pupil which fails to respond to light, but not to efforts at accommodation; absence of pupil reflex, etc.

Art'efacts. Artificial structures or productions. From *ars*, art; *factus*, made.

Asta'sia. Inability to stand. From *a*, privative, and *ιστημι*, to stand.

Asta'sia aba'sia. A condition of defective automatic co-ordination, impairing the power of standing or walking. From *ἀ*, privative, and *ἱστημί*, to stand; *ἀ*, privative, and *βασις*, step.

Aste'rion. Point of meeting of the lambdoid, parieto-mastoid, and occipito-mastoid sutures in the skull. From *aster*, a star.

Astheno'pia. Weakness of sight. From *ἀσθενὴς*, weak, and *ὤψ*, the eye.

Atax'ia or *At'axy.* Disorder: applied especially to muscular inco-ordination. From *ἀ*, privative, and *τάξις*, order.

Ath'etoid move'ments. Irregular movements, such as are seen in athetosis. The term *athetoid* is applied to a form of chorea characterized by slow, gradual, worm-like movements of the fingers and toes. From *ἀθετος*, without fixed position.

Atheto'sis. Inability to retain the fingers and toes in any one position. From *ἄθετος*, without fixed position, from *ἀ*, privative, and *τίθημι*, to place.

At'rophy. Decrease in size with corresponding loss of power. From *ἀ*, privative, and *τροφη*, nourishment.

Au'ra. A sensation like that of a vapor or emanation preceding an epileptic or hysterical seizure. From *aura*, a vapor.

Bacil'li. Micro-organisms, made up of cylindrical or oval cells, which, when connected, form rods or cells, many of which are pathogenic. From *baculus*, a rod; *bacillus*, a small rod.

Bacte'ria. A genus of Schizomycetes, which include micro-organisms that were at one time classed with the infusoria. They include micrococci, sarcinæ, bacteria proper, bacilli, vibriones, spirochætæ, and spirilla. From *βακτηριον*, a small stick.

Base'dow's disease. Exophthalmic goitre. From Von Basedow, who described it.

Bell's pal'sy or *paral'ysis.* Paralysis of the facial nerve. From Sir Charles Bell, who described it.

Belonepho'bia. Morbid dread of pins and needles. From *βελονη*, a pin, and *φοβος*, fear.

Bleph'arospasm. Spasm of the orbicularis palpebrarum muscle. From *βλεφαρον*, eyelid, and *σπασμος*, spasm.

Brachycephal'ic. Having a short head. From *βραχυς*, short, and *κεφαλη*, head.

Breg'ma. Point of junction of the coronal and sagittal sutures. From *βρεχω*, to moisten.

Bul'bar paral'ysis. Motor paralysis, with muscular atrophy, affecting the motor cranial nerves and attached muscles, especially the hypoglossal nerve, and certain filaments of the facial and of the spinal accessory. From *bulbus*, medulla oblongata, and *paralysis*.

Cais'son disease. A series of symptoms dependent upon working in compressed air, as in a caisson or diving-bell. Sometimes called *diver's paralysis*.

Cal'car a'vis. The hippocampus minor. Lat., a bird's spur.

Cal'carine. Pertaining to the hippocampus minor; as *calcarine fissure*, a groove on the occipital lobe corresponding to the hippocampus minor. From *calcar*, a spur; *calcar avis*, the hippocampus minor.

Carpholog'ia. The generally delirious action of gathering flocculi ("picking the bedclothes"), significant of extreme cerebral irritability, in low fevers, etc. From *καρφος*, flocculus, and *λεγω*, to gather.

Cat'alepsy. State of suspended consciousness and volition, with rigidity of voluntary muscles. From *κατὰ*, down, and *λαμβάνω*, to fall, to seize.

Cath'ode. Negative pole or electrode of a galvanic battery. From *κατα*, downward, and *ὁδος*, a way.

Caud'ate nu'cleus. That portion of the corpus striatum which is within the ventricle; the lenticular nucleus forming the other portion. From *cauda*, a tail.

Cellulif'ugal cur'rents. A term applied to nervous currents which flow out of the cell through the axis-cylinder processes. From *cellula*, a small cell, and *fugio*, to flee.

Cellulip'etal cur'rents. A term applied to nervous currents which flow into the cell through the protoplasmic processes. From *cellula*, a small cell, and *peto*, to seek.

Cen'tres. The chief or central points of physiological activity, the functions of which can usually be localized.

Centrif'ugal fi'bres. Efferent fibres, which pass from a centre to convey impressions to the periphery. From *centrum*, centre, and *fugio*, to flee.

Centrip'etal fibres. Nervous fibres passing to a centre, as afferent or sensory fibres. From *centrum*, centre, and *peto*, to seek.

Cen'trum ova'le. The oval medullary portion seen when the hemispheres of the brain are sliced away. From *centrum*, centre, and *ovale*, oval.

Cerebel'lum. That portion of the medullary mass contained in the cranial cavity which occupies the lower occipital fossæ below the tentorium. From *cerebrum*, brain; *cerebellum*, little brain.

Cer'ebro-spi'nal. Relating to the brain and spinal cord. From *cerebrum*, brain, and *spina*, spine.

Chias'ma. A crossing or decussation of certain nerve-fibres; *e. g.*, *Chiasma opticum*, the crossing of fibres of the optic nerve.

Chor'da tym'pani. A filament of the Vidian nerve, which enters the tympanum. From *chorda*, cord, and *tympanum*, the drum of the ear.

Chore'a. A condition attended with irregular involuntary clonic contractions of the muscles, one form being known as St. Vitus's dance. From *χορεία*, a dance.

Chorema'nia or *Choreoma'nia.* Chorea, or St. Vitus's dance, attended with convulsive emotions in hysterical or neurotic individuals.

Cinc'ture-feel'ing. A feeling as if the waist was encircled by a tight girdle or belt, noticed in certain nervous affections. From *cinctura*, a girdle.

Cing'ulum. A bundle of fibres lying in the gyrus fornicatus. Lat., a girdle or zone.

Claustropho'bia. Fear of enclosed places. From Lat., *claustrum*, an enclosed place, and *φόβος*, fear.

Claus'trum. A layer of gray matter situate between the island of Reil and the external capsule. From *claustrum*, a barrier (*claudo*, to shut).

Claw-hand. A condition seen in progressive muscular atrophy, due to sinking of the interosseous spaces on the back of the hand and incomplete extension of the terminal phalanges, with contraction of the antagonistic muscles.

Clethropho'bia. Fear of enclosed or fastened places. From *κλῆθρον*, a bar, bolt, and *φόβος*, fear.

Clon'ic. A term employed to designate certain irregular convulsive movements, in which relaxation alternates with spasm. From *κλονος*, tumultuous action.

Coch'lea. Anterior portion of the internal ear; its winding form suggesting resemblance to a spiral shell. From *κοχλίας*, a snail with a spiral shell.

Co'ma. A profound lethargy. From *κῶμα*, a swoon, deep sleep.

Com'missures. Bands or cords of nervous matter connecting different important portions of the cerebrum, cerebellum, etc. From *committo*, to join together.

Contrac'ture. Persistent and wax-like contraction of a muscle. From *con*, together, and *traho*, *tractum*, to draw.

Convolu'tions. Smooth tortuous eminences on the surface of the cerebral hemispheres. From *convolvo*, to entwine.

Co-ordina'tion. The working in harmony of muscles in the performance of any function. From *con*, together, and *ordino*, to put in order.

Coprola'lia. Involuntary use of obscene words, a symptom in some forms of insanity. From *κοπρὸς*, ordure, filth, and *λαλέω*, to talk or prate.

Cor'pora amyla'cea. Microscopic starchlike bodies found in the brain and spinal marrow. From *corpora*, bodies, and *amylum*, starch.

Cor'pora mammilla'ria. Mammillary bodies or eminences, forming the bulbs of the fornix. From *mammilla*, a small breast; diminutive of *mamma*, a breast.

Cor'pus callo'sum. A white medullary band seen on separating the two hemispheres of the brain, which it connects with each other. From *corpus*, body, and *callosus*, hard.

Cor'pus stria'tum. Anterior cerebral ganglion, forming a portion of the floor of the lateral ventricle, and now known of two distinct masses of gray matter, the caudate and the lenticular nucleus. Literally, striated body.

Cor'tex. The cortical or gray substance of the brain, seen on the external parts of the cerebrum and cerebellum. From *cortex*, bark.

Cre'tinism. A form of idiocy especially prevalent in the deep valleys of certain mountainous regions of Europe; commonly accompanied by an enormous goitre. The name is thought to be derived from "Chrétien," Christian, as implying freedom from moral responsibility—sinlessness.

Cri'ses. Violent attacks of pain in the gastric, laryngeal, or iliac regions, simulating organic disease of those parts; characteristic of locomotor ataxia.

Cru'ra. The peduncles of the cerebrum and cerebellum. From *crura*, legs (from their leglike appearance).

Cu′neus. Cuneate or wedge-shaped lobule on the surface of the occipital lobe of the brain. From *cuneus*, a wedge.

Cyrtom′eter. An instrument for measuring the curve of any portion of the body. From κυρτος, convex, μέτρον, measure.

Decussa′tion. An x-shaped crossing of nerve-fibres or filaments, as of the optic nerve. From *decussatio*, a crossing.

Demen′tia. Loss of reasoning power, or incoherence of ideas. From Lat., *de*, out of, and *mens*, the mind.

Dendrit′ic. Tree-shaped, tufted, villous. From δένδρον, a tree.

Dichot′omous. Dividing into two branches of the same or nearly equal size; bifurcating. From διχοτομεω, to cut into halves.

Diple′gia. Double symmetrical paralysis. From δίς, twice; πληγὴ, a stroke, a blow.

Diplo′pia. Double vision, two objects being seen instead of one; due to derangement of the axes of vision. From δεπλοος, twofold, and οψις, vision.

Di′ver's paral′ysis. See *Caisson disease.*

Dolichocephal′ic. Having a long head, the antero-posterior diameter being excessive. From δολιχος, long, and κεφαλη, head.

Dor′sad. Toward the dorsal aspect. From *dorsum*, back.

Dynamom′eter. Instrument for measuring the force of muscular contractions. From δύναμις, power, and μέτρον, a measure.

Dysæsthe′sia. Obscured, diminished, or even abolished sensation, particularly of the special senses. From δυς, difficult, and αισθησις, sensation.

Dys′trophy. Imperfect or defective nutrition. *Progressive muscular dystrophy* is a synonym of progressive muscular atrophy. From δυς, with difficulty, and τροφη, nourishment.

Echola′lia. A symptom of palmus, consisting of the repetition of words heard. From ἠχὼ, echo, and λαλέω, to talk or prate.

Ecs′tasy. An entranced condition with visions, the body being meanwhile in a cataleptic condition. "Out of one's self." From ἐκ, out of, and στάσις, station.

Elec′trodes. Poles of a galvanic battery. From *electricity*, and ὁδος, a way.

Electrol′ysis. Decomposition through the influence of the electric current. From *electricity*, and λυσις, solution.

Elec′tro-therapeu′tics or *-ther′apy.* Treatment of disease by electricity. From *electricity*, and θεραπευω, to cure.

Em′bolism. Plugging of a vessel with an embolus (plug) separated from the interior of the heart or a bloodvessel, and carried along in the circulating blood until it produces obstruction, as of a cerebral artery.

Encephali′tis. Inflammation of the brain (encephalon). From ἐγκέφαλος, the brain, and *itis*, a suffix denoting inflammation.

End-bask′ets. Thick and wavy terminal ramifications, having the appearance of baskets, surrounding the cells of Purkinje.

End-bulbs. Terminal nerve-corpuscles in the lips, tongue, palate, etc.

Ep′ilepsy. A cerebro-spinal disease in which the patient suddenly falls, with loss of consciousness and convulsive movements of the muscles. From ἐπὶ, upon, and λαμβάνω, to fall, to seize.

Er′ethism. Augmentation of the vital phenomena, through irritation, in any disease or organ. From ἐρεθίζω, to irritate.

Er′gotism. A condition resulting from the excessive use or abuse of ergot or spurred rye; of which vertigo, convulsions, torpor, paralysis, and dry gangrene may be symptoms.

Erythromelal′gia. A nervous disease characterized by a circular patch of redness and swelling, with pain in the part affected, usually the feet. From ἐρυθρος, red, μελος, a limb, and ἀλγος, pain.

Exophor′ia. A tending of the visual lines outward. From ἐξ, out of, and φορέω, to carry.

Exophthal′mic goi′tre. A disease characterized by cardiac palpitation, with rapid pulse; enlargement of the thyroid gland, and prominence of the eyes or exophthalmus. From ἐξ, out of, and ὀφθαλμὸς, eye.

Exophthal′mus. Abnormal protrusion of the eye. From ἐξ, out of, and ὀφθαλμὸς, eye.

Farad′ic. Relating to faradization, or electricity by induction, *Faradism.* From Faraday, the celebrated chemical and physical investigator.

Fenes′tra ova′lis. Aperture in the inner tympanum. Lat., oval window.

Formica′tion. An itchy or creeping sensation, as if ants were crawling over the surface of the body. From *formica*, an ant.

Fornica′tus. Arched, as the gyrus fornicatus or fornicate convolution, immediately above the corpus callosum. From *fornix*, an arch.

For′nix (arch or vault). Medullary body in the cerebrum, on the middle line, beneath the corpus callosum and above the middle ventricle.

Foudroy′ant (Fr.). Sudden, overwhelming.

Fried′reich's disease. A hereditary disease, consisting of nystagmus, ataxia, defective speech, loss of knee-jerk, and curvature of the spinal column. Named from its describer.

Fu′ror transito′rius. An outbreak of violent fury, lasting for a few hours, and terminating in deep sleep, from which the patient awakes without the slightest memory of its occurrence, and never recurring in the same person. From *furor*, fury, and *transitorius*, transient.

Fur′uncle. A boil. Lat., furunculus.

Gang′lia. Enlargement or knots in the course of a nerve, usually belonging to the great sympathetic system. Plural of *ganglion*, a knot.

Genic′ulate bod′ies. Eminences situate at the inferior and external portion of the optic thalami. From *geniculum*, a small knee or joint.

Gi′gantism. Extraordinary size. From γιγας, a giant.

Gir′dle-feel′ing. A feeling as if the waist was encircled by a tight girdle or belt, noticed in certain affections of the nervous system.

Glabel′la. The part of the face between the eyebrows. From *glaber*, smooth.

Glio′sis. Sclerosis with excavated spaces in the gray substance of the brain, giving rise to atrophy of the optic nerves and olfactory bulbs, and other symptoms resembling general paralysis. From γλιος, glue, and *osis*, suffix denoting morbid condition.

Glo′bus hyster′icus. A symptom of hysteria, being a sensation as if a ball was present in the throat. From *globus*, ball.

Glos'so-la'bio-larynge'al paral'ysis. A motor paralysis, with muscular atrophy, affecting the motor cranial nerves and attached muscles, especially the hypoglossal nerve, certain filaments of the facial and of the spinal accessory. From γλωσσα, tongue, *labium*, lip, and λαρυγξ, larynx.

Glos'so-pharynge'al. Relating to the tongue and pharynx; as the *glosso-pharyngeal nerve*, which is distributed to those organs and others in the vicinity.

Grand mal (Fr.). "Great disease:" applied in epilepsy to the fully developed fit.

Graves's disease. Exophthalmic goitre. From Dr. Graves, its describer, in 1835.

Gum'ma. A syphilitic gummy elastic tumor formed in the periosteum, met with in all the important viscera. From *gummi*, gum.

Gy'rus. Literally, a ring or circle. Used in anatomy of the nervous system to designate any one of certain convolutions of the brain.

Hæmatomyel'ia. Spinal hemorrhage. (For definition, see *Hæmatorrhachis.*) From ἁιμα, blood, and μυελος, marrow.

Hæmator'rhachis. An effusion of blood about, within, or between the membranes of the spinal cord, or into the substance of the cord. From ἁιμα, blood, and ῥαχις, the spine.

Hebephre'nia. So-called insanity of the period of puberty. From ἥβη, puberty, and φρην, the mind.

Heb'etude. Dulness of intellect or of sense, sometimes noticed in brain affections. From *hebes*, dull.

Helicomon'ades. The micro-organisms of syphilis. Discovered by Klebs in 1878. From ἑλιξ, a spiral, and μονας, a mound.

Hemianæsthe'sia. Loss of sensation in the lateral half of the body, caused by lesions of various portions of the central nervous system. From ἡμι, half, αν, privative, and αισθησις, sensation.

Hemianop'sia. A deficiency of one-half of the visual field. The term is applied to the crossing of the rays of light in the media and front of the retina. From ἡμι, half, α, privative, and ὢψ, the eye.

Hemio'pia. A deficiency of one-half of the visual field. The term is applied to the condition of the retina. From ἡμι, half, οπτομαι, to see.

Hemiat'rophy. Atrophy of one side of the body from impaired nutrition. From ἡμι, half, α, privative, and τροφη, nourishment.

Hemicran'ia. Headache of one side of the head. From ἡμι, half, and κρανιον, skull.

Hemiple'gia. Paralysis affecting one-half of the body. From ἡμι, half, and πληγὴ, a stroke or blow.

Hem'ispheres. Literally, half-spheres. A term applied to the upper spheroidal portion of the brain. From ἡμι, half, and σφαιρα, sphere.

Hippocam'pal. Relating to the hippocampus; as the *hippocampal fissure.* From ἱππος, a horse, and καμπος, a sea-monster.

Hippocam'pus. Name given to two elevations in the ventricles of the brain, the *hippocampus major* and *minor.* From ἱππος, horse, and καμπος, a sea-monster.

Hunt'ington's chore'a. A form of hereditary chorea in adults, the symp-

toms of which are peculiar dancing and postural movements and grimaces, mental impairment and muscular inco-ordination.

Hy′aline. Glassy or transparent. From ὕαλος, glass.

Hydroceph′alus. A collection of fluid within the brain; dropsy of the head. From ὕδωρ, water, and κεφαλή, head.

Hydromyel′ia. Fluid in the cavities of the spinal cord. From ὕδωρ, water, and μυελὸς, marrow.

Hydropho′bia. The aggregate of symptoms produced by infection from the virus communicated by the bite of an animal affected with rabies; excessive dread of water and difficulty of deglutition being prominent. See *Rabies.* From ὕδωρ, water, and φόβος, fear.

Hyperacu′sis. Unusual acuteness or sensitiveness of hearing. From ὑπερ, over, and ἀκουσις, hearing.

Hyperœ′mia. Abnormal accumulation of blood in the capillaries. From ὑπερ, over, and αἱμα, blood.

Hyperidro′sis. Excessive sweating. From ὑπερ, excessive, and ἱδρως, sweat.

Hy′pertrophy. Overgrowth from superabundant nutrition, with deficiency of waste. From ὑπὲρ, over, and τροφή, nourishment.

Hyp′notism. An artificial condition of suspended sensation, consciousness, and will-power, produced, by various methods, by another person, as by suggestion, so called. From ὕπνος, sleep.

Hypochon′dria. Morbid fears; formerly supposed to have their origin in the region of the hypochondrium.

Hypoglos′sus. The hypoglossal nerve, distributed to muscles connected with the tongue and hyoid bone. From ὑπο, under, and γλωσσα, tongue.

Hyste′ria. A morbid emotional condition reputed to have its seat in the uterus. From ὑστέρα, the uterus.

Hys′tero-ep′ilepsy. Hysteria associated with epileptiform convulsions and other nervous symptoms.

Inco-ordina′tion. Lack of correspondence between mental manifestations and muscular action, as in locomotor ataxia, etc. From *in*, not; *con*, together; *ordino*, to put in order.

Infundib′ulum. Literally, a funnel A funnel-shaped collection of gray matter attached to the pituitary body. From *in*, in, and *fundo*, to pour.

Inhibit′ion. Action of restraining or prohibiting, as of nervous influence to muscles to which such nerves are supplied. From *inhibeo*, to restrain.

In′ion ("occiput"). The occiput or the occipital protuberance.

Insom′nia. Sleeplessness. From *in*, not, and *somnus*, sleep.

In′sula cer′ebri. The island of Reil, in the brain. *Insular* signifies relating to the isle of Reil. From *insula*, island, and *cerebrum*, brain.

Inter′calate. To insert; to place between. The term *Intercala′tum* is applied to the locus niger. From *inter*, between, and *calo*, to place.

Intracra′nial. Within the cavity of the skull, as *intracranial syphilis*, *intracranial hemorrhage*, etc. From *intra*, within, and κρανιον, skull.

Jaw jerk. A motion produced by tapping the jaw while the mouth is open; it is diminished or absent in some nervous diseases.

Katatonʹia. A form of insanity characterized by mania, melancholia, and cataleptic symptoms, manifested in alternation. From κατὰ (intensive), and τόνος, tension.

Kenophoʹbia. Same as *Agoraphobia.* Fear of open spaces. From κενὸς, hollow, empty, and φόβος, fear.

Knee-jerk. Motion produced by tapping the patellar tendon while the leg is placed loosely over the opposite knee; increased or diminished in nervous diseases.

Lagophthalʹmus. A partial or complete paralysis of the orbicular muscle of the eyelid, preventing closure. From λαγως, the hare, and ὀφθαλμος, the eye.

Lambʹda. The junction of the lambdoidal and sagittal sutures, a point from which measurements are taken in craniological examinations. From the Greek letter Δ.

Lanʹdry's paralʹysis. Acute ascending paralysis, characterized by motor, or almost entirely motor, paralysis of the lower extremities, then of the trunk, upper extremities, neck, and face. Described by Landry in 1859.

Larʹvated. Hidden or obscure: applied by Falret to a form of epilepsy without *petit mal*, or in which it is very slight; *epilepsic larvê* (Fr.). From Lat., *larva*, a mask.

Lemnisʹcus. A bundle of fibres in the pons and crus cerebri, appearing superficially as a triangular band, winding obliquely around the superior peduncles from the funiculus olivarius to the corpora quadrigemina. Lat., a ribbon.

Lenticʹular. Having the shape of a lens or lentil, as the *lenticular ganglion.* From *lens*, a lentil; *lenticula*, a small lentil.

Lepʹto-meningiʹtis. Inflammation of the pia mater. From λεπτὸς, thin, slender, and *itis*, suffix denoting inflammation.

Leucocythœʹmia. Increase of leucocytes with decrease of red corpuscles in the blood. From λευκὸς, white, κύτος, cell, and αἷμα, blood.

Limʹbic lobe. A lobe which surrounds the corpus callosum. From *limbus*, a border.

Lingʹula. A thin lobule of gray matter derived from the anterior border of the cerebellum and blended with the valve of Vieussens. Diminutive of *lingua*, tongue.

Lithœʹmia. The presence of uric acid (sometimes called lithic acid) in the blood. From λίθος, stone, and αἷμα, blood.

Localizaʹtion. Specification of a certain part or structure as a centre of physiological functions or processes, or of pathological actions. From *locus*, a place.

Locomoʹtor ataxʹia. A chronic morbid condition of the spinal cord, attended with loss of power of co-ordination of voluntary muscular movements, lightning-pains, absent tendon-reflex, etc. From *locus*, a place, and *moveo*, *motum*, to move; *a*, privative, and ταξις, order.

Loʹcus cœruʹleus. A dark-brown or bluish spot in the floor of the fourth ventricle; also, a collection of large, dark, pigmented cells in the region behind the posterior quadrigeminal bodies. From *locus*, place, and *cœruleus*, blue.

Lordo'sis. Curvature of bones generally, but especially of the vertebral column forward, sometimes due to paralysis of the muscles of the spine or of the abdomen. From λορδώσις, curvature.

Ma'nia. A form of aberration of mind characterized by acute mental excitement and exaggerated nervous action. From μανία, madness.

Mar'ie's malady. Acromegaly. After Marie, who first described it.

Mas'ochism (Krafft-Ebing). A sexual perversion in which a member of one sex takes pleasure in being dominated by the other, as when the male has sensuous delight in submitting to humiliating and degrading acts from the female. From Sacher-Masoch, a novelist who wrote on this subject.

Massage (Fr.). Friction and manipulation of the body. From Fr., *masser*, to shampoo.

Masseur (Fr.). A man who practises massage.

Masseuse (Fr.). A woman who practises massage.

Mas'toid. Nipple-shaped. The projection on the temporal bone behind the ear is called the *mastoid process*. From μαστὸς, nipple, and εἶδος, like.

Medul'la oblonga'ta. The prolongation of the spinal cord upward into the cranium. From *medulla*, marrow, and *oblongatus*, prolonged.

Med'ullated fi'bres. Nerve-fibres covered with a medullary sheath. From *medulla*, marrow.

Megacephal'ic. See *Mesocephalic.* From μέγας, great, and κεφαλη, head.

Melanchol'ia. Melancholy; supposed by the ancients to be caused by black bile. From μέλας, black, and χολή, bile.

Ménière's disease. A complex of symptoms, of which vertigo is the chief, occurring with lesions of the external, middle, or internal ear. After Ménière, who first described it.

Meningi'tis. Inflammation of the meninges of the brain and cord. From μῆνιγξ, membrane, especially that enveloping the brain and cord; the pia mater; and *itis*, suffix denoting inflammation.

Menin'go-encephali'tis. Inflammation of the brain and its membranes. From μηνιγξ, membrane, ἐγκεφαλος, brain, and *itis*, suffix denoting inflammation.

Menin'go-myeli'tis. Inflammation of the spinal cord and its membranes. From μηνιγξ, membrane, μυελος, marrow, and *itis*, suffix denoting inflammation.

Mes'merism. A form of hypnotism. From the name of its originator, Mesmer.

Mesocephal'ic. Term employed to designate a skull whose capacity is between 1350 c.cm. and 1450 c.cm. (being determined by filling the skull with shot poured into it through the foramen magnum). If less than 1350, the skull is said to be *microcephalic;* if over 1450, *megacephalic*. From μεσος, middle, and κεφαλη, head.

Mi'crobe. Microbes are minute unicellular organisms (schizomycetes). The class includes the micrococcus, bacillus, spirillum, etc., commonly known as bacteria. From μικρὸς, small, and βίος, life.

Microcephal'ic. See *Mesocephalic.* From μικρος, small, and κεφαλη, head.

Migraine (Fr.). Pain in the temporal or orbital region of one side of the head; sick headache. Abridged from Hemicrania. From ἡμι, half, and κρανίον, skull.

Mil'iary an'eurism. Aneurism of the smallest arteries, of the cerebellum especially. From *milium*, millet, and ἀνευρυσμα, dilatation.

Milliam′père. A measure of electricity, one-thousandth part of an ampère. From *mille*, a thousand, and *ampère*, unit of strength of electro-magnetic current shown by force of one volt through one ohm. From the name of a French scientist.

Milliampèreme′ters. Instrument for measuring minute electricity, as by milliampères (which see). From *milliampère*, and *μέτρον*, measure.

Monoma′nia. A form of mental aberration formerly supposed to be confined to one subject of delusion. From *μονος*, alone, and *μανια*, madness.

Monople′gia or *monople′gic paral′ysis.* Paralysis limited to one organ or part, caused by circumscribed lesions. From *μονος*, single, and *πληγη*, stroke.

Morphinoma′nia or *Morphioma′nia.* A morbid craving for morphine. Mania from the use of morphine.

Morvan's disease. A disease commencing with neuralgia, which is followed by paresis and analgesia, finally by one or more whitlows, with necrosis of the phalanges. From Morvan, who described it in 1883.

Myeli′tis. Inflammation of the spinal cord. From *μυελὸς*, marrow, and *itis*, suffix denoting inflammation.

Myoät′rophy. Atrophy of a muscle. From *μυς*, muscle, *a*, privative, and *τροφη*, nourishment.

Myopath′ic at′rophy. Progressive muscular atrophy. From *μυς*, muscle, and *παθος*, disease; *a*, privative, and *τροφη*, nourishment.

Myosclerot′ic paral′ysis. A chronic disease, usually called pseudo-muscular hypertrophy or progressive muscular sclerosis, causing paraplegia and great increase in the size of the limbs. From *μυς*, muscle, and *σκληρος*, hard.

Myo′sis. Contraction of the pupil. From *μύω*, to close the eyes, and *osis*, suffix denoting morbid condition.

Myoton′ia. Thomsen's disease. Painless tonic contraction of voluntary muscles, lasting several seconds at a time, and thus interfering with locomotion and other movements. From *μῦς*, muscle, and *τόνος*, tension.

Mysopho′bia. A morbid fear of being unclean. From *μυσος*, uncleanliness, and *φοβος*, fear.

Myxœde′ma. Gelatinous infiltration of the skin, usually accompanied by dulness of the senses, slow monotonous speech, and cretinoid aspect. From *μύξα*, mucus, and *οἴδημα*, a swelling.

Na′sion. Craniometrically the median point of the naso-frontal suture. From *nasus*, the nose.

Necroph′ilism. Insane sexual desire for a corpse. From *νεκρὸς*, a corpse, and *φιλέω*, to love.

Ne′oplasms. New formations or growths, as tumors. From *νεος*, new, and *πλασμα*, growth.

Neural′gia. Pain felt over the distribution of a nerve, usually without fever. From *νεῦρον*, a nerve, and *ἀλγος*, pain.

Neurasthe′nia. Nervous exhaustion. From *νεῦρον*, a nerve, and *ἀσθένεια*, weakness.

Neuri′tis. Inflammation of a nerve. From *νεῦρον*, a nerve, and *itis*, suffix denoting inflammation.

Neurog′lia. A reticular substance lying between the ganglionic cells and nerve-fibres in the brain and cord. From *νεῦρον*, a nerve, and *γλία*, bird-lime, glue.

Neuro'ma. A tumor composed of nerve-tissue. From νεῦρον, a nerve, and *oma*, suffix denoting tumor.

Neu'ron. A nerve. The cerebro-spinal cord. From the Greek, νεῦρον, a nerve.

Neuro'sis. Nervous disease without specified lesions. From νεῦρον, a nerve, and *osis*, suffix denoting morbid condition.

Neurot'omy. Section or division of a nerve. From νεῦρον, nerve, and τομη, incision.

Night-ter'rors. A sudden attack of terror in sleeping children, as if of some impending danger.

Notomyeli'tis, Inflammation of the spinal cord. From νωτος, back, and μυελος, marrow.

Nu'cleus. The deep origin of a nerve; a group of cells forming central termination of a nerve or bundle of nerve-fibres. From *nucleus*, a kernel.

Nystag'mus. Involuntary rapid movements of the eyeball. From νυσταγμὸς, nodding of the head, slumber.

Oinoma'nia. Dipsomania; passion for wine. From οἶνος, wine, and μανία, madness.

Ol'ivary bodies. Oval protuberances on the lateral surface of the medulla oblongata. From *oliva*, an olive.

Oper'culum. Literally, a lid or cover. A triangular eminence covering a considerable portion of the island of Reil.

Ophthalmople'gia. Paralysis of ocular muscles. From ὀφθαλμὸς, eye, and πληγη, stroke.

Oti'tis. Inflammation of the ear. From οὖς, ear, and *itis*, suffix denoting inflammation.

Pachymeningi'tis. Inflammation of the dura mater, its chronic form causing thickening of this membrane. From παχὺς, coarse, thick, μῆνιγξ, membrane, and *itis*, suffix denoting inflammation.

Pain'ful points. Points on a neuralgic surface, particularly sensitive to pressure.

Pal'mus (adj. *palmo'dic*). "The twitches." Convulsive tic. A nervous affection characterized by twitching, by involuntary imitation and readiness to perform any act directed, however objectionable, and by a tendency to the use of indecent words. From παλμός, twitching, palpitation.

Papilli'tis. Inflammation of the papilla or optic disk. From *papilla*, a nipple, the optic disk, and *itis*, suffix denoting inflammation.

Paracen'tral lob'ule. Area between the calloso-marginal and paracentral sulci on the median surface of the cerebral hemispheres. From παρα, beyond, and κεντρον, centre.

Paræsthe'sia. Morbid perversion of sensibility, as in numbness, etc. From παρα, beyond, and αισθησις, sensation.

Paral'ysis. Impairment or complete loss of power of motion or of sensation, or of both. It may be general or local. From παρα, beyond, and λυω, to loosen.

Paramyoc'lonus. Convulsive tremor in symmetrical muscles, the spasms ceasing during sleep. From παρὰ, beside, beyond, μῦς, muscle, and κλόνος, disturbance.

Paramyoton'ia. Tonic muscular spasm. From παρὰ, beside, beyond, μῦς, a muscle, and τονος, tension.

Paranoi'a. Unsoundness of mind; crankiness. From παράνοια, fatuity, madness (παρὰ, beside, beyond, and νοῦς, the mind).

Parapha'sia. Aphasia of conduction. Substitution of words which do not express the meaning intended to be conveyed. From παρὰ, beside, beyond, and ἀφασια, aphasia.

Paraple'gia. Paralysis of the lower extremities. From παρὰ, beside, beyond, and πληγη, stroke.

Par'esis. Impairment of motor power. Partial paralysis. Sometimes used synonymously with general paralysis. From παριημι, to relax.

Park'inson's disease. Paralysis agitans, or shaking palsy. After Parkinson, who first described it in 1817.

Par vag'um. The pneumogastric nerve. From *par*, pair, and *vagus*, wandering.

Pathognomon'ic. Characteristic of the disease. From παθος, disease, and γιγνωσκω, to know.

Pe'duncles. Mainly used to denote the three cerebellar processes, the superior, the middle, and the lower, connecting the cerebellum with other portions of the brain. From Lat., *pedunculus*, a small foot.

Perimys'ium. The areolar tissue of striated muscle. From περι, around, and μυς, a muscle.

Periph'eral. Relating to the periphery or circumference, as peripheral nerves. From περιφερεια, circumference.

Phreni'tis. Inflammation of the brain or its membranes, or the series of morbid symptoms associated with it. From φρην, mind, and *itis*, suffix denoting inflammation.

Petit mal (Fr.). "Slight disease:" applied in epilepsy to the initial symptoms of the fit.

Pe'trous. Hard; resembling stone; as petrous portion of temporal bone. From πέτρα, a rock.

Pluricord'onal cells. A variety of many-column cells of the spinal cord, having long axis-cylinders. From *plus*, many, and (Fr.) *cordon*, cord.

Pneumogas'tric. Relating to the lungs and stomach. The pneumogastric nerve is so called because it is distributed chiefly to the organs of the chest and abdomen. From πνευμων, lung, and γαστηρ, stomach.

Pol'io-encephali'tis. Acute inflammation of the cortex, affecting chiefly the motor region. From πολιὸς, hoary, gray, ἐγκέφαλος, the brain, and *itis*, suffix denoting inflammation.

Poliomyeli'tis. Inflammation of the gray matter of the spinal cord. From πολιὸς, hoary, gray, μύελος, marrow, and *itis*, suffix denoting inflammation.

Polyæsthe'sia. Abnormal sensibility, in which a touch impressed at a single point is felt at two or more points. From πολυς, many, and αισθησις, sensation.

Pons Varo'lii. An eminence at the upper portion of the medulla oblongata and at the base of the brain. First described by Varolius. From *pons*, a bridge.

Porenceph'aly. A defect of structure consisting of abnormal passages in, or absence of, any part of the cerebrum. From πόρος, a passing over, bridge, and ἐγκέφαλος, the brain.

Præcen'tral. In front of the centre. A term applied to a special convolution and fissure of the brain. From *præ*, before, and *centrum*, centre.

Præcu'neus. A quadrate lobule on the median surface of the cerebral hemisphere, in front of the cuneus. From *præ*, in front of, and *cuneus*, a wedge.

Pro'cess. A prolongation or eminence. From Lat., *processus*, a going forward.

Prosopal'gia, Prosoposal'gia. Facial neuralgia. From πρόσωπον, the face, and ἄλγος, pain.

Pro'toplasm. A substance common to all organisms and essential to all the phenomena of life. It is semi-translucent, viscous, and without visible structure. From πρῶτος, first, and πλάσσω, to mould.

Pseu'do-mus'cular hy'pertrophy. A chronic disease causing paraplegia and great increase in size of the lower limbs, and occasionally of the upper limbs. From ψευδης, false, *musculus*, muscle, ὑπερ, over, and τροφη, nourishment.

Psych'ical cells. Name given by Cajal to certain cortical cells. From ψυχη, the soul or mind.

Pter'ion. In craniotomy the region in which the extremity of the great wing of the sphenoid approaches the frontal, parietal, and temporal bones. From πτερον, a feather or wing.

Pto'sis. Drooping of the upper eyelid, from paralysis of the levator palpebræ muscle or induration of the eyelid. From πτωσις, a falling.

Pul'vinar. The posterior eminence of the optic thalamus. From *pulvinus*, a cushion.

Quadrigem'inal bodies. Four rounded eminences under the corpus callosum; the optic lobes. From *quadrigeminus*, fourfold.

Ra'bies. Canine madness, or hydrophobia of animals; hydrophobia being rabies manifesting itself in the human being. From Lat., *rabies*, madness or fury.

Raph'e. (More correctly *rhaphe.*) A line, ridge, or seam in the middle line. From ῥαφη, a suture.

Raynaud''s disease. An affection consisting of paroxysmal attacks of pain, pallor, coldness, numbness, and anæsthesia of the lower extremities, and a gangrenous condition of the toes. After Raynaud, who described it.

Reac'tion of degenera'tion. Alteration in the response of a nerve and muscle to the galvanic and faradic current, caused by lesion of such nerve and muscle.

Re'flexes. Reflections by an efferent nerve of impressions that have been carried to a nervous centre by an afferent nerve. From *re*, back, and *flecto*, *flexum*, to bend.

Reinforce'ment. A term applied to augmentation of reflex response, when muscular action or stimulation operates at the same time on different parts from those embraced in the reflex circle.

Rest'iform bod'ies. Two peduncles or medullary projections from each side of the upper extremity of the medulla oblongata. From *restis*, a rope, and *forma*, shape.

Rhachialgi'tis. A badly compounded word (literally, inflammation of pain

in the spine), denoting what is sometimes called "spinal irritation." From ῥαχις, spine, ἀλγος, pain, and *itis*, suffix denoting inflammation.

Rhe'ophore. Any substance conducting the electric current; usually the electrodes held by, or applied to, the patient. From ῥεος, current, and φοζεω, to carry.

Rhe'ostat. An instrument which regulates the current of electricity or measures its resistance. From ῥεος, a current, and ἱστημι, to stand.

Rom'berg's sign or *symp'tom.* Uncertain, swaying gait, in the dark or with the eyes closed. After Romberg, who first described it.

Sadis'mus. A condition that is the opposite of *Masochismus*, which see. In sadismus a member of one sex takes sensual delight in exercising force upon one of the other sex. From the name of the Marquis de Sade, a prurient writer.

Sagit'tal. A term applied to a groove, sinus, suture, etc., of the skull, from the arrow-shaped appearance. From *sagitta*, an arrow.

Saltato'ric. Of a jumping, jerky, or dancing character. From Lat., *saltator*, a dancer.

Sclero'sis. Induration or hardening of tissues. From σκληρὸς, hard.

Somnam'bulism. Sleep-walking. From Lat., *somnus*, sleep, and *ambulo*, to walk.

Spasm. Involuntary muscular contraction; a convulsion. From σπασμος, act of drawing.

Spasmod'ic. Relating to or characterized by spasm, as *spasmodic diseases*. From σπασμος, act of drawing.

Spas'tic. Relating to contraction. From σπαστικὸς, contracted.

Sphyg'mograph. Instrument for automatically recording the rate and force of the pulse. From σφυγμος, pulsation, and γράφω, to write.

Spini'tis. Inflammation of the spinal cord; myelitis. A hybrid word, from *spina*, the spine, and *itis*, suffix denoting inflammation.

Sta'tus epilep'ticus. A condition, sometimes fatal, in epilepsy, when one fit follows another in rapid, continuous, and regular succession. From *status*, state, and ἐπìλαμβανω, to seize upon.

Stepha'nion.. Point at which the coronal sutures and temporal lines cross. From στεφανη, the crown.

Stri'æ acus'ticæ. Transverse white lines on the inferior portion of the floor of the fourth ventricle, which are associated with the roots of the auditory nerve.

Substan'tia ferrugin'ea. A collection of large, dark, pigmented cells on the fasciculi teretes of the fourth ventricle of the brain. From *substantia*, substance, and *ferrum*, iron.

Substan'tia ni'gra. A tract of cerebral gray matter between the crusta and tegmentum, consisting of numerous pigmented nerve-cells of irregular shape. From *substantia*, substance, and *nigra*, dark.

Subthal'amic. Under the optic thalamus, as the *subthalamic body*.

Syph'ilis. A specific infectious venereal disease. Perhaps from σιφλόω, to debase, humble, disgrace; some authorities consider the derivation to be from σῦς, swine, and φιλέω, to love.

Syphilo'ma. A syphilitic tumor, as a gumma or syphilitic gummy tumor of the brain. From *syphilis*, and *oma*, suffix denoting tumor.

Syringomyel'ia. See *Syringomyelitis.*

Syringomyeli'tis. The formation of a fissure or cavity in the cord. From σῦριγξ, a hollow tube, pipe, μυελὸς, marrow, and *itis*, suffix denoting inflammation.

Ta'bes. A wasting away; emaciation. From Lat., *tabeo*, to waste away.

Ta'bes dorsa'lis. Literally, dorsal wasting; a synonym for progressive locomotor ataxia. From *tabeo*, to waste away, and *dorsum*, back.

Tachycar'dia. Abnormal rapidity of the heart's action. From ταχυς, quick, and καρδια, the heart.

Tegmen'tum. The postero-superior portion of the crura cerebri. Lat., a covering.

Telangiec'tasis. Dilatation of the extremities of bloodvessels or of the capillaries. From τελος, the end, ἀγγειον, a vessel, and ἐκτασις, extension.

Ten'don-re'flex. Reflex contraction of a muscle, caused by percussion of its tendon. See *Reflex.*

Tetanil'la (diminutive of *tetanus*). Tonic, bilateral, intercurrent, usually painful spasms of certain muscular groups, without loss of consciousness. From τέτανος, distention, convulsion, cramp.

Tet'anus. A disease characterized by rigidity, with paroxysms of tonic convulsions; lockjaw. From τέτανος, distention, convulsion, cramp.

Tet'any. See *Tetanilla.*

Thal'ami op'tici. Two masses of nervous matter at the base of the brain, from which the optic nerves have a partial origin. From θαλαμος, bed, and οπτικος, relating to sight.

Ther'mo-æsthesiom'eter. A registering instrument with which to test the temperature-sense. From θερμη, heat, αισθησις, sensation, and μετρον, measure.

Thermom'eter, cer'ebral. Instrument to determine and register the temperature of different regions of the skull. From θερμη, heat, μετρον, measure, and *cerebrum*, brain.

Thom'sen's disease. See *Myotonia.* From Dr. J. Thomsen, of Schleswig, who, in 1876, first described it in his own case.

Thrombo'sis. Obstruction of any portion of the circulatory apparatus, as of the brain, by a coagulum. From *thrombus*, a clot.

Tic (Fr.). Spasmodic action; twitching.

Tic douloureux. (Literally, painful tic.) Painful twitching from neuralgia of branches of the fifth pair of nerves.

Tituba'tion. A reeling, stumbling, staggering gait, as in cerebellar disease. From *titubo*, to stumble or totter.

Ton'ic spasms. Rigid muscular contractions without relaxation. From τονος, tension.

Topoäl'gia. A name given to pain in different regions which are not anatomically or physiologically delimited. From τοπος, a place, and ἀλγος, pain.

Traum'atism. The condition of the organism produced by a wound or injury. From τραυμα, a wound.

Trigem'inus, or *Trigem'inal nerve.* The fifth pair of nerves, which divides into three branches. From *tri*, in composition three, and *gemini*, double; triplets.

Tris'mus. Lockjaw: a spastic closure of the lower jaw. From τρισμος, gnashing of the teeth.

Troph'ic. Relating to nutrition. From τροφή, nourishment.

Trophoneuro'sis. Failure in nutrition due to impairment of nervous action. From τροφή, nourishment, and νευρον, a nerve.

Typhoma'nia. A disease characterized by furious outbreaks of maniacal excitement and violence, with intermissions. Also, condition of lethargy and delirium in typhus. From τυφος, stupor, and μανια, madness.

Un'cinate or *Uncina'tus* (Lat.). Hooklike, hooked, furnished with hooks, as uncinate convolution or gyrus of the brain, which terminates in a hooklike process. From *uncus*, a hook.

Vag'abond pains. Wandering pains, of a stabbing and fulgurant kind, symptomatic of locomotor ataxia.

Vag'us. See *Par vagum.*

Ve'lum interpos'itum. A vascular membrane reflected from the pia mater into the interior of the cerebrum through the transverse fissure. From *velum*, a veil, and *interpositus*, placed between.

Ve'lum medulla're. A term variously applied to the valve of Vieussens, the medullary strip associating the medulla and the cerebellum, and the small posterior valve of the cerebellum. From *velum*, a veil, and *medulla*, marrow.

Ven'trad. Toward the belly; anterior to the vertebral column. From *venter*, belly, and *ad*, to.

Ven'tricles. Five cavities in the interior of the brain, known as the two lateral, the middle, the fourth, and the fifth ventricles. From *ventriculus*, a small belly.

Verbigera'tion. The repetition by an insane person of one word or sentence or sound, but without sense or sequence. From *verbigero*, to talk.

Ver'mes. Two medullary projections at the surface of the cerebellum, the vermiform processes. From *vermis*, a worm.

Ver'tebral. Of or pertaining to a vertebra. Having vertebræ. Vertebrate. From *vertebra*, a spinal bone (*verto*, to turn).

Ver'tigo. Dizziness. From Lat., *verto*, to turn.

Ver'tigo, au'ral. See *Ménière's disease.* From *verto*, to turn, and *auris*, the ear.

Waller'ian law. A law that a nerve degenerates in the direction in which the impulse is normally conveyed by it.

Wrist-drop. Paralysis of the muscles of the forearm, induced by lead-poisoning.

Wry-neck. Contraction of the muscles of the region of the neck, generally of one side, causing a fixed position of the head on that side.

INDEX.